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THE EFFECT OF PYLORIC OBSTRUCTION IN RELATION TO GASTRIC TETANY*

By W. G. MacCallum, Joseph Lintz, H. N. Vermilye, T. H. Leggett, and E. Boas

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The nature of gastric tetany has long been a matter of interest and the theories already advanced to account for it are quite unsatisfactory. According to one it is due to desiccation of the tissues, whereas another ascribes it to the absorption of toxic materials from the stagnating contents of the dilated stomach. It is known that if a communication be established between the stomach and the intestine by a gastro-enterostomy so that the contents of the stomach can once more pass into the intestine, the symptoms disappear at once. It was with the desire of determining the nature of the changes produced by pyloric obstruction that the following experiments were carried out. But, although they give clear evidence that a certain chain of events follows such an obstruction, they do not necessarily give an explanation of gastric tetany in the human being, and we have had no opportunity to make the same observations on man.

It was observed long ago (1909) by one of us (MacCallum) that when the pylorus was completely obstructed and the stomach frequently washed out, an animal wasted rapidly and died in a few days, usually with violent convulsions which were not precisely of the same character as the twitchings seen in parathyroid tetany.

* These experiments were begun in 1909 in Baltimore, where all the effects of pyloric obstruction and of the administration of chlorides and hydrochloric acid were observed. They were resumed later with more accurate quantitative measurements in New York.

They were described, practically as they now appear, at the meeting of the American Society for Experimental Pathology held in New York with the Federation of Biological Associations at Cornell University Medical School in January, 1917, but various circumstances connected with my leaving New York for Baltimore and the press of work during the war have delayed their publication up to the present. In the meanwhile a paper by W. S. McCann has appeared in the *Journal of Biological Chemistry*, 1918, xxxv, 553, which describes quite similar experiments and results but with a somewhat different interpretation. (W. G. M.)

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We held the idea at that time that, since nothing was absorbed from the stomach, while water was given abundantly through the intestine, the older explanations offered for gastric tetany were faulty and that the convulsions must be due to loss of hydrochloric acid in the gastric juice. Later experiments have supported this view and we have tried to work out the nature of the whole disturbance.

Although many different methods have been employed from time to time in the attempt to obstruct the pylorus partially or completely we finally returned to the simplest, which consists in cutting through the stomach just above the pylorus and closing it off with sutures so that it becomes a blind sac on the end of the œsophagus. The pyloric end with the duodenum was then brought into the abdominal wound and sutured there. Through it food and water were given, but in the later experiments, in order to eliminate all intake of chlorides and to prevent loss of bile and intestinal contents, this opening was closed except for a tube through which distilled water was introduced. The food which could be given in this way always contained chlorides and when it was given the symptoms following pyloric obstruction appeared only slowly so that the animal lived about a week. When nothing but water was given, convulsions appeared in about 48 hours and death soon followed. Such a result could not be due to starvation, because a dog will live a long time without food.

It is unfortunate that our observations upon the daily intake and output of chlorides are not more complete, but they are sufficient to show that under the conditions of the operation, even when no chlorides are given in the food, the obstructed stomach continues to secrete hydrochloric acid. Estimation of the chlorides in the stomach washings show that a considerable amount is lost to the body and this must be sufficient, in the absence of any intake, to lower the chlorides of the body rapidly. Indeed it is difficult to devise any other way in which such an abstraction of chlorides could be attained since the tissues ordinarily hold tenaciously to their chloride content when the intake is decreased. It seems that the gastric mucosa can still exercise its function of secreting hydrochloric acid even when the plasma chlorides are diminished.

6709. Pylorus obstructed May 6, 1909. Animal fed through intestinal fistula with sugar and beef together with distilled water. Six days later, May 12, violent twitching and stiffness, flexor spasm of fore feet and rigid clinching of jaws. Toward the end of the experiment sodium chloride was injected intravenously and relieved the twitching. Chlorides estimated in the washings from the stomach as follows:

6709 Chlorides as NaCl	May							
	6	7	8	9	10	11	12	13
Gastric fluid	...	1.56	3.0	3.3	.42	...	...	...
Urine	...	.27	.56	.22	1.6	.65	1.9	...

6909. Pylorus obstructed May 18, 1909. Animal fed as above with washed beef, sugar and water. Well until May 23, when there was violent twitching of all the muscles. Was given calcium lactate intravenously which diminished the twitching.

6909 Chlorides as NaCl	May					
	18	19	20	21	22	23
Gastric fluid	....	5.37	4.8	2.7	2 6	....
Urine	....	....	2.2	1.6	.72	3.2

6309. Pylorus obstructed April 22, 1909. Fed with capsules of dried washed beef (chloride free) and water. Slight twitching beginning April 27. Was given sodium chloride intravenously. Died late next night.

6309 Chlorides as NaCl	April					
	22	23	24	25	26	27
Gastric fluid	....	....	1.7	3.07	3.1	1.4
Urine	....	....	2.7	.6	....	....

These observations show that the loss of chlorides from the stomach was continued for days after the operation. The excretion in the urine tended to decrease day by day after the operation. Later when we had found that the analysis of the blood plasma showed more directly and more precisely the changes in the chlorides, we made these determinations every day and ceased to analyze stomach washings and excreta for chlorides.

Since the chloride lost in the gastric juice is in the form of free hydrochloric acid, it seemed probable that the sodium ion would be retained in the circulating fluids and that the alkali reserve of the blood might be increased.

In the next series of experiments, therefore, we studied the changes in the plasma chlorides, the alkali reserve as determined by Van Slyke's method and the electrical excitability of the nerves.

In brief we found that, especially in those cases in which no chlorides were given by the intestinal fistula, the recognizable chlorides in the plasma dropped rapidly. The average course taken from 12 cases was as follows, beginning with the day of the operation: 6.6, 6.4, 5.1, 4.6, 3.9, but in some cases the chloride content of the plasma fell to 2.5 or 2.8.

The carbon dioxide combining power was found to rise as the chlorides decreased. Averaging once more eight cases in which this is tabulated we find that the change proceeds as follows, beginning with the day of operation: 46.2, 42.4, 55.2, 61.1, 66.6, 71.9, 74.7, 80 (volume in c. c. per 100 c. c. blood). It is hardly possible to average the records of the electrical excitability, but an approximation was made in six cases by adding the figures KC, KO, AC and AO into one figure for each observation. Then if these figures be averaged for the series of animals beginning with the day of operation they run as follows: 7.6, 7.1, 4.5, 2.8, in which of course the lowest number represents the lowest current necessary and therefore the highest excitability.

The following are illustrative protocols:

1605. Pylorus obstructed October 30, 1916. Animal fed through the intestinal fistula with milk and eggs; developed distinct tetany-like twitchings on November 6, followed by violent convulsions and death. During this time the dog's weight sank from 7630 grams to 6210 grams. Electrical excitability was very distinctly heightened. The plasma chlorides sank.

1605		October		November					
		30	31	1	2	3	4	5	6
		Operation						Tetany	
Electrical excitability	KC	....	0.2	....	0.2	...	0.05	....	0.05
	KO	....	Neg. 5	....	Neg. 5	...	Neg. 5	....	1.2
	AC	....	1.0	....	0.4	...	0.4	....	0.4
	AO	....	1.0	....	1.2	...	1.2	....	0.4
Plasma chlorides		....	....	6.41	5.67	...	5.67	....	4.10

1609. Pylorus obstructed November 24, 1916. Fed through fistula with milk and eggs. On November 27, distinct tetanic twitchings were developed with high electrical excitability. An intravenous injection of 35 c. c. of 10% NaCl was given which relieved the twitchings but these reappeared the next day and the dog died.

1609		November								
		23	24	25	26	27			28	
			Pyloric obstruction			NaCl ↓				
Electrical excitability	KC	0.3	....	0.2	...	0.15	0.3	0.4	0.3	
	KO	Neg. 5	....	Neg. 5	...	1.4	3.6	2.2	1.8	
	AC	1.2	....	0.8	...	0.8	0.7	1.3	0.8	
	AO	1.4	....	1.6	...	0.4	1.2	2.5	1.2	
Plasma chlorides		7.4	....	....	5.43	4 9	6.4	5.8	5.3	
Total NaCl intake		1.20	1.24	1.31	1.33	3.95		...	Neg. Bal.	
Total NaCl output		2.11	1.30	5.33	2.81	2.92		...	5.44	

1612. After a preliminary operation to establish an intestinal fistula on Dec. 2, 1916, the pylorus was obstructed on December 8. The dog lived until Dec. 14. There was nothing resembling tetany but observations on the chlorides and the alkali reserve are recorded. The animal was fed with milk, eggs and sugar.

1612		December							
		7	8	9	10	11	12	13	14
Electrical excitability	KC	...	0.2	0.3	0.3	0.2	0.3	0.3	0.2
	KO	...	Neg. 5	Neg. 5	Neg. 5	Neg. 5	Neg. 5	Neg. 5	3.0
	AC	...	1.2	1.4	1.4	1.0	1.2	1.2	1.0
	AO	...	2.6	2.8	2.4	2.0	2.0	1.8	1.0
Plasma chlorides		6.6	6.7	6.8	5.7	5.4	4.7	4.3	2.5
CO_2 combining power (vols. in c. c. per 100 c. c. of blood)		...	49.0	58.6	62.4	61.4	63.3	74.9	80
Total NaCl intake		1.43	1.27	1.34	1.28	1.1	1.1	1.06	...
Total NaCl output		1.48	0.75	3.09	0.2	1.48	....	2.14	...

1618. Pyloric obstruction December 14, 1916. Given distilled water only through intestinal fistula; developed distinct and continuous twitchings on December 16 and died.

1618		December			
		14	15	16	
Electrical excitability	KC	....	0.2	0.2	0.2
	KO	....	Neg. 5	Neg. 5	1.8
	AC	....	0.8	1.0	0.6
	AO	....	1.4	1.8	1.0
Plasma chlorides		6.6	6.7	5.5	4.4
CO_2 combining power		46.7	39.1	43.0	33.1

1620. Pylorus obstructed December 17, 1916. Dog given distilled water only through intestine. Violent convulsion with opisthotonos on December 19. Electrical excitability greatly increased, plasma chlorides diminished. Alkali reserve not greatly heightened. Given 50 c. c. of 1% HCl intravenously. This lowered the alkali reserve but scarcely affected the electrical excitability. Died about four hours later.

1620		December					
		17	18	19			
				Convulsion		HCl ↓	
Electrical excitability	KC	0.2	0.2	0.2	0.1	0.3	0.4
	KO	Neg. 5	Neg. 5	1.0	1.2	1.6	1.8
	AC	1.0	1.0	0.8	0.6	0.6	1.2
	AO	1.4	0.4	0.8	1.2	0.8	0.8
Plasma chlorides		....	7.2	5.63	5.22	...	...
CO_2 combining power		4.15	38.2	57.3	50.7	47.5	...

1621. Pylorus obstructed December 18, 1916. Given only distilled water through intestinal fistula. On December 20 extremely weak and apathetic. No twitching but occasional convulsive stretching into a position of opisthotonos. Given 50 c. c. 2 M. NaCl intravenously. Much improved and lived for three days longer with gradual reproduction of the same condition.

1621		December							
		18	19	20			21	22	23
				NaCl injected		↓			
Electrical excitability	KC	...	0.1	0.1	0.1	0.2	0.1	0.1	...
	KO	...	Neg. 5	1.6	2.2	5.0	2.4	4.4	...
	AC	...	1.0	0.4	0.4	1.6	0.8	0.8	...
	AO	...	1.0	1.2	1.0	1.0	1.2	1.6	...
Plasma chlorides		6.9	7.7	5.8	3.8	6.0	5.6	4.5	4.2
CO_2 combining power		43.4	41.4	49.3	64.5	61.2	61.7	50.7	74.7

1622. Pylorus obstructed December 18, 1916. Animal given only distilled water through the intestinal fistula. The dog lived five days during which the plasma chlorides became greatly diminished and the alkali reserve increased. An attempt to introduce $\frac{N}{10}$ HCl in dilute solution ended fatally.

1622		December						
		18	19	20	21	22	23	
							$\frac{N}{10}$ HCl ↓	
Electrical excitability	KC	....	0.1	0.1	0.3	0.3	0.1	0.3
	KO	....	Neg. 5	5.0	Neg. 5	Neg. 5	Neg. 5	Neg. 5
	AC	....	1.0	1.2	1.5	1.6	1.0	1.6
	AO	....	2.4	1.6	2.6	2.8	2.0	2.8
Plasma chlorides		7.03	6.99	....	4.48	3.54	2.79	....
CO_2 combining power		32.1	35.7	53.2	71.1	78.0	78.4	67.3

1704. Pylorus obstructed January 20, 1917, and animal given only distilled water through the intestinal fistula. The electrical excitability rose and on Janury 22 the dog had a violent convulsion lasting one minute. An hour later was given 50 c. c. 2 M. NaCl intravenously. Electrical excitability remained high but the dog was quiet and fairly well. On January 26, 50 c. c. 2 M. NaCl were again given intravenously. The dog remained alive until January 29 and there was no further twitching.

1704		January									
		20	21	22	23	24	25	26	27	28	29
				NaCl				NaCl			
Electrical excitability	KC	....	....	0.05 ↓ 0.10	0.05	0.05	0.05	0.2 ↓ 0.05	0.1	0.1	....
	KO	....	....	1.6 Neg. 5	1.6	2.8	2.8	2.6 Neg. 5	Neg. 5	2.8	....
	AC	....	....	0.5 1.0	0.4	0.8	0.8	0.6 1.2	0.8	0.2	....
	AO	....	....	0.9 1.6	1.0	1.2	1.0	0.8 1.6	1.0	1.0	....
Plasma chlorides		6.1	5.7	4.1 4.1	4.6	4.6	3.6	2.5 4.1	3.7	....	....
CO_2 combining power		41.7	38.1	43.0 44.0	47.3	68.6	69.1	82.7 70.0	71.5	94.	....

1705. Pylorus obstructed January 21, 1917. Given only distilled water by intestinal fistula. No symptoms on January 22 and 23, but on the morning of January 24 the dog was found in convulsions or "tetany." This was slower and more deliberate, less clonic than parathyroid tetany. There were no fibrillary tremors of the tongue. The convulsions were severe, stretching the animal into opisthotonos with attempts at vomiting. They were rather more like the convulsions of ammonia poisoning than those of parathyroid tetany. On January 24, after the convulsion had lasted four hours, the animal was given 50 c. c. 2 M. NaCl solution intravenously. The convulsions disappeared and the animal lived until January 27 without any further attack.

1705		January						
		21	22	23	24	25	26	27
					NaCl			
Electrical excitability	KC	....	0.2		0.2 ↓ 0.6	0.6	0.4	0.4
	KO	....	Neg. 5	0.05	Neg. 5 Neg. 5	Neg. 5	Neg. 5	3.8
	AC	....	1.2	Neg. 5	1.4 1.6	1.6	1.0	0.8
	AO	....	2.2	1.0	2.6 2 8	4.0	2.0	1.2
Plasma chlorides		5.7	5.6	2.2	3.2	4.0	3.2	3.0
CO_2 combining power		53.7	43.	4.4 47.3	....	63.5	65.8	71.5 34.5

1707. Pylorus obstructed January 26, 1917. As usual, the stomach was washed out twice daily and found to contain 300-500 c. c. of fluid. No symptoms until January 29, when there were violent convulsions. The mouth was opened wide and there were retching and salivation. No convulsions on January 30, but the animal was very stuporous and appeared to be dying. Given intravenously 100 c. c. 2 M. NaCl. January 31, very apathetic. Given 300 c. c. 2 M. NaCl intravenously, seemed rather improved but died in the afternoon. This was of course an extreme dose of sodium chloride amounting to 41 grams. It raised the plasma chlorides above the normal figure but only when the dog was moribund.

1707		January						
		26	27	28	29	30	31	
					Convulsions	NaCl	NaCl	
Electrical excitability	KC	0.4	0.4	0.1	0.1	0.2	0.2	0.6
	KO	Neg. 5	Neg. 5	Neg. 5	Neg. 5	Neg. 5	Neg. 5	4.0
	AC	1.2	2.0	1.0	0.8	0.8	1.2	1.4
	AO	2.0	3.8	2.4	2.4	2.3	3.0	1.4
Plasma chlorides		6.2	6.1	5.2	4.5	3.6	4.7	10.0
CO_2 combining power		60.0	45.0	58.0	64.0	74.9	65.3	24.2

1714. Pylorus obstructed March 6, 1917. Given only distilled water by intestinal fistula. No symptoms until March 9 when violent convulsions with much frothing at the mouth occurred. In this case practically no change in electrical excitability was observed, although characteristic alterations in the plasma chlorides and alkali were present.

1714		March			
		6	7	8	9
Electrical excitability	KC	0.3	0.2	0.2	0.2
	KO	Neg. 5	Neg. 5	Neg. 5	Neg. 5
	AC	1.2	1.0	0.7	1.0
	AO	1.8	1.6	1.0	1.8
Plasma chlorides		7.4	5.4	5.0	4.1
CO_2 combining power		46.0	48.0	60.0	72.0

From these experiments it is seen that a peculiar condition accompanied by convulsions appears whenever the pylorus is obstructed so that the acid gastric juice is all removed and no chlorides are given in the food. It must be recognized that the condition is not the same as that produced by parathyroidectomy. The muscular rigidity with vibrating clonic twitchings and extreme tachypnœa are lacking. Instead the rather apathetic animal usually lies quiet until seized with a violent universal convulsion which throws the body into extreme and rigid opisthotonos with attempts at vomiting and abundant salivation. After this is over he sinks into a kind of coma with slow, deep respirations. Rapid respirations of great volume often precede the onset of the convulsion. The electrical excitability is heightened definitely and up to the time of the convulsions it increases but never to a point comparable with that seen in parathyroid tetany, and even during the most violent twitching, stiffness and clenching of the jaws the KO reaches only 1.8 or 1.6, whereas in parathyroid tetany it is not unusual to have it 0.6 or 0.4.

The rapid diminution in the plasma chlorides with the corresponding rise in the alkali reserve as measured by van Slyke's method was constant. We, therefore, thought that the condition might be relieved by the replacement of the chlorides, and as a matter of fact injections of sodium chloride into the blood stream when the symptoms were well developed regularly caused the disappearance of the convulsions and a general improvement in the condition, and lowered for a time the electrical excitability.

When a dog otherwise treated in the same way, that is by obstruction of the pylorus and washing out of the stomach, was given distilled water with the addition of 10 grams of sodium chloride daily, it lived for a week or more without symptoms, maintained a constant proportion of plasma chlorides and a constant alkaline reserve as measured by the carbon dioxide combining power. Moreover, the electrical excitability of the nerves remained normal.

1711. Pylorus obstructed February 22, 1917. Given distilled water with 10 grams NaCl through intestinal fistula. Remained quiet; no twitching or convulsions. Walked about apparently well until February 27 when it was found dead. The wound had been torn open and there was an acute pleuritis.

1711		February.					
		22	23	24	25	26	27
Electrical excitability	KC	0.2	0.2	0.1	0.4	0.8	....
	KO	Neg. 5	Neg. 5	Neg. 5	Neg. 5	Neg. 5	....
	AC	1.2	0.8	0.7	1.2	2.2	....
	AO	1.2	0.8	0.8	1.2	2.8	....
Plasma chlorides		6.8	6.6	6.4	7.0	8.0	....
CO_2 combining power		43.8	50.4	52.2	50.1	49.0	....

1712. Pylorus obstructed February 26. Given distilled water with sodium chloride as before. Remained well until March 2 when he was apathetic and there were attempts at vomiting. Died on March 4 without infection or other obvious cause of death.

1712		February			March			
		26	27	28	1	2	3	4
Electrical excitability	KC	0.1	0.1	0.1	0.1	0.3	0.6	....
	KO	Neg. 5	Neg. 5	Neg. 5	Neg. 5	Neg. 5	Neg. 5	....
	AC	0.8	0.8	0.8	0.6	1.6	2.2	....
	AO	1.2	1.6	2.2	1.8	2.6	Neg 5	....
Plasma chlorides		6.7	7.0	7.0	7.5	8.2	8.2	8.3
CO_2 combining power		35.0	49.0	46.2	49.2	30.9	42.8	35.0

We had of course regarded the whole alteration as due to loss of hydrochloric acid with the result that an excess of base had been left in the circulating fluids, so that the natural experiment would be the replacement of the chlorine ion by the introduction of hydrochloric acid. This was done several times but without much success (1620, 1622, 1709 and others), whereas the introduction of sodium chloride has a life saving effect and the administration of sodium chloride continuously after the operation prevented the development of changes in the electrical excitability, disturbances of the alkali reserves or symptoms. The fate of the Na ion requires explanation since it appears that so long as the Cl ion is supplied there is no heightened alkali reserve.

The prompt effect of calcium salts in stopping parathyroid tetany led to their trial in some of these cases. The injection of calcium lactate, although it exerts a temporary effect in stopping the twitchings or modifying the convulsions, has no such controlling influence, as is seen in the tetany following parathyroidectomy after which an animal can be kept perfectly well for a long time by the proper administration of calcium.

We had started with the idea that the loss of chlorides might throw out of function soluble calcium in the plasma or even actually precipitate it and we made numerous analyses of the blood to determine the changes in the proportion of calcium. The analyses were made (not to show the absolute, but the relative, amount of calcium in the plasma) by dialyzing 10 c. c. of blood received in 1 c. c. of 15% sodium citrate solution for 24 hours at 4° C. in a fish bladder sac against 100 c. c. of a solution containing 0.8% of sodium chloride and 0.3% sodium citrate. The clear colorless dialysate was boiled, acidulated and precipitated with oxalate after the method of McCrudden. Titrations were made against $\frac{N}{100}$ potassium permanganate. Controls were made by estimating the calcium in the residue in the sac and by analyzing the dialysate after known amounts of calcium chloride had been added to the blood and they show that a proportional amount of calcium appears in the dialysate. All that we venture to say from these studies is that there appears to be no characteristic change in the proportion of calcium in the blood from the time before the operation until the death of the animal. (As for the method, although it is probably open to many objections, it has the advantage of eliminating many difficulties in the analysis of blood for calcium and of giving accurate proportional results, which was all we required.)

It is clear, however, that there is a distinct change in the acid base equilibrium in favor of the alkali reserve and it seemed possible that this might be reproduced temporarily by the injection of sodium carbonate or bicarbonate which was done with the following results:

1614. Blood studied before injection: From 4.20 p. m. to 5.10 p. m. a gradual injection was made of 2 M. Na_2CO_3, 100 c. c. in all being run into the vein. The electrical excitability became greatly heightened temporarily. No convulsions were produced and there were no further effects.

1614		December 21							
		4	4.20	4.40	5	5.20	5.40	6	6.20
Electrical excitability	KC	0.1	...	0.05	...	0.05	...	...	0.1
	KO	Neg. 5	...	Neg. 5	...	1.8	...	...	Neg. 5
	AC	0.6	...	0.6	...	0.4	...	...	0.6
	AO	1.0	...	1.4	...	0.6	...	...	1.4
Plasma chlorides		6.8	...	....	...	6.4	...	...	7.2
CO_2 combining power		50.4	...	....	...	119.6	...	...	74.0

1623. Blood studied before injection. From 4.50 to 5.15, 160 c. c. 2 M. Na_2CO_3 run into the vein. No marked symptoms, recovery prompt.

1623		December 22						
		4.20	5.20	6.20	7.20	8.20	9.20	10.20
		Na_2CO_3						
Electrical excitability	KC	0.2	0.1	0.1	...	...	...	0.1
	KO	Neg. 5	0.8	1.0	...	...	...	Neg. 5
	AC	0.8	0.5	0.6	...	...	...	1.2
	AO	1.4	0.2	0.4	...	...	...	1.2
CO_2 combining power		47.0	138.6	...	...	...	...	....

1624. After normal electric reactions had been determined the injection of 10% Na_2CO_3 into a vein was begun at 4 p. m. and continued to 5 p. m., 240 c. c. being run in. The animal became stiff with twitching and fibrillation of the tongue. Electrical excitability much heightened. Recovery complete.

1624		January 6				
		3.30	4.00	4.30	5.00	5.30
				Na_2OC_3		
Electrical excitability	KC	0.5	....	0.3	0.4	0.2
	KO	Neg. 5	....	...	2.6	1.0
	AC	1.6	....	1.6	2.4	1.0
	AO	1.6	....	0.6	0.6	0.4
CO_2 combining power		....	....	...	...	...

1701. Injection of 2 M. Na_2CO_3 begun at 3 and continued to 4.15 p. m. during which time 150 c. c. were run in. After this the

electrical reactions disappeared and no contraction could be elicited. At 3.45 dog became stiff and stretched out. At 4 p. m. a violent convulsion occurred with twitching of the whole body.

1701		November 30							
		2.30	2.45	3.00	3.15	3.30	3.45	4.00	4.15
			Na_2CO_3				Convulsions		Na_2CO_3
Electrical excitability	KC	...	0.1	...	0.2	0.1	0.5	0.5	Neg.
	KO	...	Neg. 5	...	Neg. 5	Neg. 5	Neg. 5	Neg. 5	Neg.
	AC	...	0.6	...	0.6	0.6	0.6	0.6	Neg.
	AO	...	0.9	...	0.5	0.3	0.2	0.4	Neg.

1702. Injection of 1 M. $NaHCO_3$ begun at 3.15 and continued until 4.30 p. m., when 250 c. c. had been run in. Vomiting at 3.50. Generalized twitchings at 4.10. Slight convulsion at 4.30. Prolonged and continuous convulsions at 4.50, after which all electric responses failed. Died at 5.15.

1702		December 1									
		3	3.15	3.30	3.45	4	4.15	4.30	4.45	5	5.15
		$NaHCO_3$						Convulsions			
Electrical excitability	KC	0.4	....	0.2	0.3	0.2	0.2	0.2	....	Neg.	Died
	KO	Neg. 5	....	Neg. 5	Neg. 5	Neg. 5	Neg.5	Neg.5	....	Neg.	..
	AC	1.4	....	1.5	1.5	0.9	1.0	0.8	....	Neg.	..
	AO	2.8	....	1.7	1.0	0.8	1.0	0.7	....	Neg.	..

1704. Three c. c. per kilo of 1 M. Na_2CO_3 were injected intravenously every 15 minutes. Weight of animal 11.45 kilos. This was begun at 10 a. m. and continued till 12.15, at which time 340 c. c. had been injected. The dog died shortly afterward. (Observations made by the students in experimental pathology under Dr. Admont Clark.)

1704		February 7									
		10	10.15	10.30	10.45	11	11.15	11.30	11.45	12	12.15
		Na_2CO_3									
Electrical excitability	KC	0.8	....	0.6	....	0.5	....	0.6	....	0.5	0.4
	KO	Neg.5	....	Neg.5	....	Neg.5	....	Neg.5	....	1.6	1.0
	AC	1.2	....	2.0	....	2.0	....	2.2	....	1.6	1.0
	AO	2.6	....	2.4	....	1.4	....	0.9	....	1.0	0.5
CO_2 combining power		46.6	55.1	60.1	73.9	83.3	87.6	96.4	104.4	112.0	121.3
Alveolar CO_2 (Marriott)		34.	33.	38.	38.	42.	40.	43.	43.	44.	45.

From these experiments it seems that practically the same symptoms, twitching, convulsions, opisthotonos, etc., can be produced by excessive injections of sodium carbonate or bicarbonate solutions as by the removal of the chlorides. The preponderance of the alkali over the acids is on a higher plane since there has been no loss of acid, but the relations are similar. The alterations of the electrical excitability parallel with those of the alkali reserve are just the same in the two cases. In both the increase of excitability is moderate and rather irregular. Sometimes it seems not to occur, while in other cases it is very definite.

An explanation of such phenomena usually means an interpretation in terms of simpler phenomena. Here we find that convulsions and heightened electrical excitability of the nerve muscle apparatus coincide with an excess of alkali. The same results are produced by the withdrawal of acid which could possibly disturb the relations of sodium, potassium, magnesium and calcium in the body.

It is possible that the disturbed equilibrium of acids and bases in itself is the cause of the symptoms. It is well known that the buffer qualities of the blood are such as to prevent any change in the actual PH. value without extreme interference with the alkaline reserve. The addition of acids lessens the alkaline reserve, but until it is used up there is compensated acidosis. Similarly the increase in the alkali content of the blood must be resisted to maintain the normal hydrogen ion concentration until an excessive amount of alkali is added. (This is not by retention of CO_2 because the alveolar CO_2 is increased.) A great excess of alkali must ultimately produce a marked change in the PH. of the blood.

The papers of Wilson, Stearns and Janney dealing with the condition of alkalosis in tetany following parathyroidectomy state that it may develop, but later on is neutralized by the acid products formed by the muscular activities incident to tetany. This result was reached by the study of the values of the dissociation constant of oxyhæmoglobin, the alveolar CO_2 pressure and the hydrogen ion concentration of the blood. McCann confirms this by observations on two cases made by Van Slyke's method. In his first case he administered magnesium sulphate and calcium chloride and there is no way of telling the relation of the changes in alkali reserve to these procedures or to the tetany. In the second case he also removed the stomach contents at intervals, which would have its own effect. None of these writers records the chemical changes in time relation to changes in electrical excitability which are so important in the diagnosis of the condition of tetany. We ourselves in a few observations have found no change in the alkali reserve in the direction of alkalosis during tetany of parathyroid origin.

1617. Parathyroidectomy December 9, 1917. Tetany beginning December 11. On December 12, the dog was found in violent tetany. Given bicarbonate intravenously which did not relieve the tetany but appeared to add its own influence. No heightening of alkali reserve during the parathyroid tetany.

1617		December				
		9	10	11	12	
					$NaHCo_3$	
Electrical excitability	KC	....	0.05	0.05	0.05 ↓	0.05
	KO	....	Neg. 5	1.0	0.6	0.6
	AC	..	0.3	0.2	0.3	0.4
	AO	....	0.8	0.8	0.4	0.4
Plasma chlorides		....	6.9	6.8	6.78	6.38
CO_2 combining power		32.8	45.4	39.8	36.5	74.9

1705. Parathyroidectomy December 18, 1917. On December 19 the dog was well. On December 20 there was violent tetany and death at 3 p. m. There was no increase in the alkaline reserve.

1705		December		
		18	19	20
Electrical excitability	KC	0.1	0.2	0.05
	KO	Neg. 5	Neg. 5	1.4
	AC	1.2	1.0	0.4
	AO	1.8	1.2	1.0
Plasma chlorides		...	...	...
CO_2 combining power		61.7	60.7	40.5

CONCLUSIONS

When the pylorus is obstructed and the gastric juice with its hydrochloric acid is constantly removed, there ensues a decrease in the chlorine of the plasma.

There is a consequent increase in the alkali reserve which becomes extreme.

The electrical excitability of the nerves is in general heightened and there are spontaneous twitchings and in most cases violent convulsions which lead to death.

All of this can be prevented by constantly furnishing a large supply of chlorides. It is less easy to cure the condition by the administration of chlorides.

The convulsive movements are not exactly like the twitchings of the tetany of parathyroidectomy in which we have found no heightened alkali reserve, but they can be produced by the injection of sodium carbonate or bicarbonate.

Since these convulsions can be stopped or prevented by sodium chloride, it remains a problem as to what becomes of the excessive base sodium and as to the specific need of the chlorine ion. Further experiments are contemplated to settle these points.

ACUTE CHOLECYSTITIS IN CHILDREN AS A COMPLICATION OF TYPHOID FEVER

By MONT R. REID and J. C. MONTGOMERY

(From the Surgical and Pediatric Clinics of The Johns Hopkins Hospital)

Following the report by Gilbert and Girode in 1890 of the case of a patient with typhoid fever from whose gall-bladder a pure culture of the typhoid bacillus was obtained, it soon became common knowledge that the gall-bladder is quite constantly infected by the bacillus during the course of the disease and not infrequently for months or even years after recovery. Chiari, in 1894, in his routine autopsies on 22 cases of typhoid fever, recovered the typhoid bacillus from the gall-bladder in 19 cases. The experimental work of Blachstein and Welch and the routine bacteriological examinations at autopsies in Professor Welch's laboratory also afforded early proof that Gilbert and Girode's statement in regard to a single case of typhoid fever would probably be applicable to all cases during some stage of the disease. It is now a well-known fact that the gall-bladder is probably always infected during the course of the disease and that the typhoid bacillus plays an important rôle in the causation of gall-stones and of cholecystitis.

The cases of empyema of the gall-bladder or acute typhoidal cholecystitis, however, that require surgical treatment or cause death during the course of the disease are few in number. Keen, in his work on "The Surgical Complications and Sequels of Typhoid Fever," reports only 30 such cases. Since his work appeared in 1898 only a few other cases have been reported. The rarity of severe gall-bladder complications during the course of the disease is also illustrated by the report of Hölscher who made post-mortem examinations on 2000 cases of typhoid fever. Of these, five showed gross pathological changes in the gall-bladder but in only one was there a perforation. More recently A. W. Webb-Johnson, in his careful study of 2500 cases of the typhoid fevers observed during the first half of the world war, reports only one case of acute cholecystitis in which surgical measures were resorted to, although in 15 cases the symptoms and signs of acute cholecystitis were sufficiently definite to be recognized.

In children acute suppurative cholecystitis of any kind is rare. Altogether there are only about 20 reported cases.*

The recent admission to this hospital of a child with a large typhoidal empyema of the gall-bladder gave to most of us our first opportunity of observing a case of this kind, and incited in the writers the desire to study our hospital records and the literature relating to this condition in children. We will give the story of this case first, although it appears as No. 17 in the tables. The other cases that we have been fortunate enough to find have not been tabulated by any author and therefore deserve to be given in abstract form in this paper. The cases occurring in the service of Professor Halsted and filed in the records of this hospital have not hitherto been reported.

CASE 17.—The Johns Hopkins Hospital. Surg. No. 47082. A white girl of eight years came into the Pediatric Service of Professor Howland on December 20, 1918. She was admitted because of great emaciation, pain in the abdomen and a large abdominal mass. These abdominal symptoms had begun about one week before her admission and apparently at the end of a long illness which began on October 15, 1918, with high fever, delirium, headache, anorexia and some diarrhea. This illness lasted for eight weeks and during this time the child was treated at her home. Shortly before the abdominal symptoms began, the mother thinks the child had become afebrile and had begun to have a voracious appetite.

On admission to the Pediatric Clinic the patient weighed 40¼ pounds. Her temperature was 98.8° F., pulse 100, and respirations 22. She was very listless and showed an extreme grade of emaciation. There were some recent and some old furuncles about the neck and shoulders. Over the sacrum and spines of the scapulæ the skin showed a little reddening and thinning—beginning bedsores.

Further interest in the physical examination was centered entirely in the abdomen. It was asymmetrically distended, the right half and flank bulging more than the left. The skin venules were quite prominent. Abdominal respiratory movements were free. No peristaltic waves could be noted. A huge mass (outlined in the

* It is mainly from the writings of von Khautz, Wharton, Eisendrath, Osler and Mason that we have obtained these figures. See bibliography.

drawing) could be felt, and easily seen. It extended down to the level of the anterior superior spines of the ilia. Laterally and upward it was continuous with the mass in the flank and with the liver dulness. The surface of the swelling was smooth. There was no spasm of the rectus muscle. The lower and inner edge of the tumor was rounded and readily definable. The liver edge could not be felt. To some of those who examined the child the

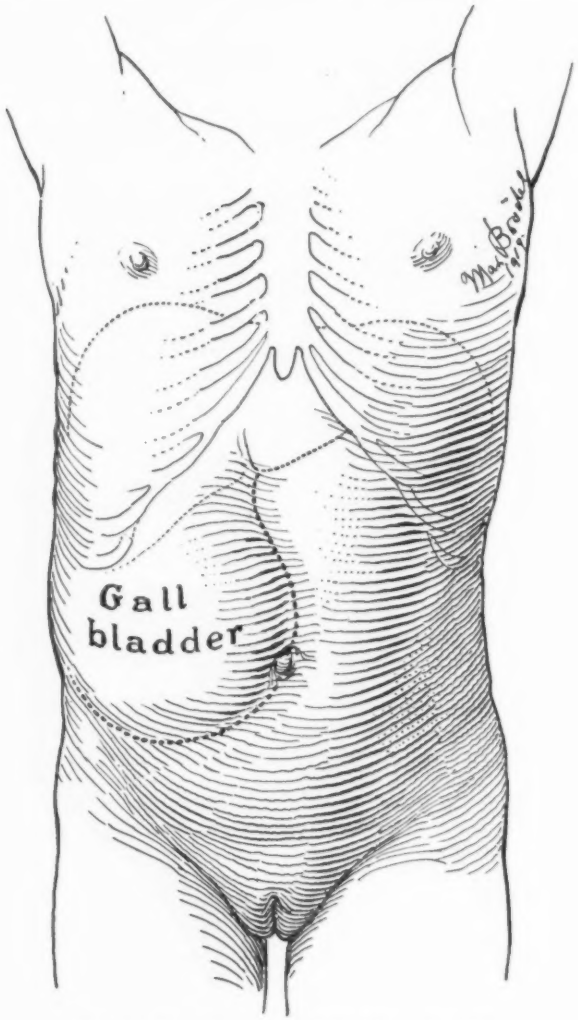

FIG. 1.—Outline of the gall-bladder. Case 17.

swelling appeared firm, elastic and possibly to be a solid tumor; to others it appeared fluctuant, particularly on bi-manual palpation through the right flank. The spleen and kidneys could not be palpated.

Laboratory Findings.—White blood count, 10,400. The vaginal smear showed no gonococci. The von Pirquet test was negative. The urine contained some albumin, and a few white blood cells. The kidneys secreted 66 per cent of phthalein in two hours. The X-Rays of the kidneys and chest showed practically nothing.

During three days' study on Dr. Howland's service the temperature varied between 99° and 104° F. No definite diagnosis was made. Retro-peritoneal sarcoma, sarcoma of the liver, cyst of the liver, abdominal abscess, appendix abscess, etc., were suggested by various men who saw her. An affection of the gall-bladder was suggested but not held to be very likely on account of the huge size of the mass.

The patient was transferred to Dr. Halsted's service and was operated upon by Dr. Reid on December 23, 1918. The seat of the trouble was found to be in the gall-bladder which was distended with between 700 c. c. and a liter of pus. The first pus to be evacuated was thin and yellowish; later it became thick and creamy. The gall-bladder was removed. No stones were present. After a moderately stormy convalescence the patient left the hospital on February 3, 1919. She gained weight rapidly and by the last of March was a normal healthy child with a slight tendency to obesity.

When the cultures from the gall-bladder showed a pure strain of *B. typhosus*, the case became clear. Previously we had believed that the swelling in the abdomen had been the sole cause of the child's illness and that it probably had begun with high fever, delirium, headache and diarrhea on October 15, 1918. We now came to the conclusion that the empyema of the gall-bladder was certainly secondary to typhoid fever and had probably developed toward the end of the disease. One week after the operation a culture from the urine showed typhoid bacilli and there was certainly a typhoidal pyelitis which disappeared before she was discharged.

The gall-bladder showed a typical picture of an acute hemorrhagic cholecystitis with necrotic areas of varying size.

CASE 1.—Husson, 1835. A child of eight years died in the third week of typhoid fever. The cause of her death was peritonitis which had developed during the course of the disease. At autopsy a perforation of the gall-bladder with resulting peritonitis was found. No stones were present.

CASE 2.—Archambault, 1852. An infant developed peritonitis in the convalescent period of typhoid fever. The autopsy showed an ulcerative cholecystitis with a perforation of the gall-bladder. The common duct was obliterated. [The presence or absence of stones is not mentioned.]

CASE 3.—Barthez and Rilliet, 1853. A child of 12 years died in the fifth week of typhoid fever. Whether the cause of death was recognized as due to a complication is not stated. The autopsy revealed a rupture of the gall-bladder with a large abscess formation about it.

CASE 4.—Chédevergne, S., 1864. This patient, aged 15, died on the fifteenth day of an illness diagnosed as typhoid fever. At autopsy an ulcerative cholecystitis was found.

CASE 5.—Stedman, C. E., 1882. A girl, aged 13 years, died on the twenty-fourth day of the disease with symptoms of peritonitis. At autopsy an ulcerative cholecystitis with a perforation of the fundus of the gall-bladder that had given rise to a general peritonitis was found. In the intestine were typical lesions of typhoid fever. No gall-stones.

CASE 6.—Stedman, C. E., 1882. A girl, aged six years, died of typhoid fever on the twenty-eighth day of the disease. The complication was diagnosed as a general peritonitis which was found at autopsy to be due to an ulcerative cholecystitis with three perforations in the fundus of the gall-bladder. The patient was a sister of the other patient whose case (No. 5) was reported by the same author.

It is interesting to note that Stedman reports the case of a third member of the same family, aged 16, who also had acute cholecystitis, but who recovered.

CASE 7.—Bond, J. W., 1886. The patient, aged 10 years, died during the course of typhoid fever from complications which were diagnosed as pneumonia and meningitis. At autopsy there was also found an empyema of the gall-bladder.

CASE 8.—Chiari, H., 1893. In the sixth week of typhoid fever the patient, aged 12 years, died apparently from pneumonia. The post-mortem examination revealed, in addition to a bronchopneumonia, a general peritonitis which was due to a ruptured

empyema of the gall-bladder. A culture from the abdomen showed *B. typhosus*.

CASE 9.—Alexieef, 1896. The patient, aged five years, developed during the third week of a well-marked case of typhoid fever the symptoms and signs of an acute cholecystitis. The gall-bladder was distended and a distinct fremitus was present over it. After this condition had persisted for about two days the child was seized with an agonizing pain, the abdomen became generally distended and the enlarged gall-bladder disappeared. These acute symptoms were probably due to a perforation of the gall-bladder, for a day or so later the operation revealed a peritonitis in the region of the gall-bladder due to a perforation of its wall. With drainage the child made a good recovery. Very carefully made cultures showed *B. typhosus*.

CASE 10.—The Johns Hopkins Hospital. Surg. No. 12174. A white boy, aged 11 years, was admitted to the Medical Service in July, 1901, with a typical case of typhoid fever. On August 5, or about the beginning of the fourth week of the disease, he began to have severe abdominal pain. He had had several attacks during the night before his transference to the surgical clinic. When seen by Dr. Mitchell, who afterward performed the operation, the physical signs were typical of an acute cholecystitis with marked distention of the gall-bladder. There was a visible tumor just to the right of the umbilicus measuring about 5 cm. in diameter. Over this there was marked tenderness and muscle spasm. The

TABLE OF CASES OF TYPHOIDAL CHOLECYSTITIS IN CHILDREN

Case	Date	Age	Time	Diagnosis	Treatment	Result	Reported by
1	1835	8	Third week.	Typhoid fever.	No operation.	Death.	Husson: Bull. Soc. anat. de Par., 1835, 104.
2	1852	Infant.	Convalescing.	Typhoid fever. Peritonitis.	No operation.	Death.	Archambault: Bull. Soc. anat. de Par., 1852, 90.
3	1853	12	Fifth week.	Typhoid fever.	No operation.	Death.	Barthez et Rilliet: Traité des maladies des enfants, Paris, 1853, 2. éd., ii, 5; 701.
4	1864	15	Fifteenth day.	Typhoid fever.	No operation.	Death.	Chédevergne: De la fièvre typhoïde et de ses manifestations. Thèse de Paris, 1864, No. 173.
5	1882	13	Twenty-fourth day.	Typhoid fever. Peritonitis.	No operation.	Death.	Stedman: Med. and Surg. Rep. Bost. City Hosp., 1882, 3. s.
6	1882	6	Twenty-eighth day.	Typhoid fever. Peritonitis.	No operation.	Death.	Stedman: Med. and Surg. Rep. Bost. City Hosp., 1882, 3. s.
7	1886	10	Not determined.	Typhoid fever. Meningitis. Pneumonia.	No operation.	Death.	Bond: Jahrb. f. Kinderh., 1886, xxiv, 291.
8	1893	12	Sixth week.	Typhoid fever. Pneumonia.	No operation.	Death.	Chiari: Prag. med. Wchnschr., 1893, xviii, 261–263, Nr. 22.
9	1896	5	Third week.	Acute cholecystitis, typhoidal.	Drainage of peritonitis.	Well.	Alexieef: Jour. Dietskaya Meditzina, 1896, No. 4; *also in* The surgical complications and sequels of typhoid fever (Keen), 1898, 268.
10	1901	11	Fourth week.	Acute cholecystitis, typhoidal.	Cholecystostomy.	Well.	Reported by the authors: Operator, Dr. J. F. Mitchell, Resident Surgeon, J. H. H., 1901.
11	1905	12	Eight months after recovery from typhoid fever.	Acute cholecystitis.	Cholecystostomy.	Well.	Reported by the authors: Operator, Dr. W. F. Sowers, Resident Surgeon, J. H. H., 1905.
12	1908	13	Convalescing.	Acute cholecystitis, typhoidal.	Cholecystostomy.	Well.	Reported by the authors: Operator, Dr. R. S. Miller, Resident Surgeon, J. H. H., 1908.
13	1908	12	Fifth week.	Typhoid fever. Intestinal perforation.	Exploratory laparotomy. Drainage of abdominal cavity.	Death.	Ashhurst: Am. J. M. Sc., Phila., 1908, cxxxv, 541.
14	1909	9	Second week.	Acute cholecystitis, typhoidal.	Cholecystostomy.	Well.	Reported by the authors: Operator, Dr. J. W. Churchman, Resident Surgeon, J. H. H., 1909.
15	1910	14	Third or fourth week.	Acute cholecystitis, typhoidal.	Cholecystostomy.	Well.	Prince: Surg., Gyn. and Obst., Chicago, 1910, xi, 416.
16	1911	15	Fifth week.	Typhoid perforation of intestine.	Cholecystostomy.	Well.	Reported by the authors: Operator, Dr. C. M. Remsen, Resident Surgeon, J. H. H., 1911.
17	1918	8	Convalescing.	Not made before the operation.	Cholecystectomy.	Well.	The authors' case.
18	1919	5	Fifth week.	Acute cholecystitis, typhoidal.	Cholecystostomy.	Well.	Deaver: Ann. Surg., Phila., 1919, lxix, 534–536.

general condition was fairly good. The leucocyte count was 10,000.

The operation was performed under cocaine anæsthesia. The gall-bladder was markedly distended and showed a definite area of beginning necrosis of its wall. The contents of the gall-bladder were a cloudy, serous-looking fluid which toward the end of the aspiration became slightly bile-stained. Under the microscope numerous clumps of bacilli were noted. In all about 300 c. c. of fluid were withdrawn. The gall-bladder was drained with rubber tubing. For a time bile was discharged freely, and when he left the hospital, six weeks after the operation, he still showed a persistent small biliary fistula. This probably closed soon after, for there is no record of the patient having had further trouble. Cultures from the gall-bladder showed *B. typhosus*. There were no stones present.

Case 11.—The Johns Hopkins Hospital. Surg. No. 17406. A white girl, aged 12 years, developed symptoms of acute cholecystitis eight months after recovery from typhoid fever. She was admitted to Dr. Halsted's service February 21, 1905. The leucocyte count was 33,000. The temperature was 102° F. and the physical signs were typical of acute infection of the gall-bladder, associated with an indefinite mass. At the operation performed by Dr. Sowers, the gall-bladder was found to contain pus, ten small gall-stones, but no bile. Cultures were not made. The stones had probably been due primarily to the typhoid bacillus but on account of the acute nature of the condition and the high leucocytosis it is not unlikely that a secondary infection had been superimposed.

This case, strictly speaking, should not come in this series. We include it because of the age of the patient, and the relatively short period between the recovery from typhoid fever and the development of gall-stones.

Case 12.—The Johns Hopkins Hospital. Surg. No. 22877. A negro girl, aged 13 years, was admitted to the medical service on July 22, 1908. The clinical picture and laboratory findings were typical of typhoid fever. On July 25, she had sharp abdominal pains accompanied by some tenderness below the umbilicus. These recurred on August 1 and at that time there was marked tenderness in the epigastric and right hypochrondriac regions, associated with a rigidity of the right rectus muscle. There was no leucocytosis. On August 15, after having had a normal temperature for a few days, she had a relapse which was accompanied by sharp abdominal pains and a temperature of 103° F. On September 4 there was marked tenderness over the gall-bladder region, pain in the right flank and rigidity of the right rectus muscle. Under ether the gall-bladder was easily felt. At the operation (by Dr. R. S. Miller) it was found to be markedly distended with a watery purulent fluid. There were no stones. The gall-bladder was drained and the child made an uneventful recovery, leaving the hospital on September 26, 1908. A culture made from the pus of the gall-bladder showed *B. typhosus*.

Case 13.—Ashhurst, A. P. C., 1908. A boy, aged 12 years, developed on the 32d day of typhoid fever, without previous symptoms, a very severe abdominal pain followed soon by the physical signs of peritonitis. A diagnosis of intestinal perforation was made and an operation was performed by Dr. G. G. Davis about five hours after the onset of the symptoms. A general peritonitis associated with a considerable amount of bile-stained fluid was found. There was no intestinal perforation. The patient was so sick that the peritoneal cavity was drained without any further attempt to discover the cause of the peritonitis. Death occurred 20 hours after the operation, and the post-mortem examination revealed a perforated gall-bladder. [There is no mention of cultures or of stones.]

Case 14.—The Johns Hopkins Hospital. Surg. No. 24693. A negro boy, aged nine years, toward the end of the second week of typhoid fever was operated upon September 20, 1909, by Dr. J. W. Churchman. The symptoms calling for surgical intervention had begun on the same day when the patient cried out with sudden severe pain in the abdomen, this pain lasting only a few minutes and then recurring frequently. The upper right half of the abdomen was tense and definitely distended. The gall-bladder could be felt. A slight difference in the two sides of the abdomen had been noted for several days before these acute attacks of pain and it was thought that a distended gall-bladder could be made out. There was an increase in muscle rigidity and definite jaundice. On account of acute attacks of pain associated with increased abdominal tenderness and a slight rise in the leucocyte count to 10,000, he was transferred to the surgical clinic.

At the time of operation the gall-bladder was distended with bile which, except for its thick consistency, appeared to be normal. Drainage with a tube was employed. The patient improved rapidly after the operation and left the hospital 54 days later. The wound was entirely healed. [There is no report of cultures.]

Case 15.—Prince, E. M., 1910. A girl, 14 years old, developed in the fourth week of typhoid fever the symptoms of an acute cholecystitis. She apparently had not had attacks of pain in the gall-bladder region previous to the day of operation when symptoms came on very rapidly. The leucocyte count was 21,000. Temperature 104° F. Pulse 130. The abdomen was very tender. There was marked spasm of the upper right rectus muscle.

At operation, a much thickened and inflamed gall-bladder was found. The contents were "clotted bile." A cholecystostomy was performed. For three days no bile was discharged but after that it flowed freely. A culture of the contents showed a "motile bacillus." Convalescence was complicated by an otitis media.

Case 16.—The Johns Hopkins Hospital. Surg. No. 26530. A negro boy, aged 15 years, during the course of typhoid fever and in the fifth week of the disease, developed signs which suggested to Dr. Remsen, who operated upon him, the probability of a perforation of the intestine. At the time of the operation the peritoneal cavity was free. The appendix showed a mild grade of inflammation which was thought to be due to a typhoid infection and the gall-bladder was markedly distended. The appendix was removed and the gall-bladder, although it did not appear particularly abnormal, was drained on account of its distention. In the gall-bladder there was thick bile but no stones. The walls of the gall-bladder itself were not necrotic. The cultures showed *B. typhosus*. The patient made an uneventful recovery and left the hospital about eight weeks after the operation.

Case 18.—Deaver, H. C., 1919. This patient, a girl of five years, was in the fifth or sixth week of typhoid fever and apparently convalescing satisfactorily when she was taken with acute abdominal pain. This was so severe and the symptoms of peritoneal irritation so pronounced that an operation was performed within two hours of the onset.

A markedly congested gall-bladder with a necrotic area in its fundus was found. No perforation had occurred. The contents were a saniopurulent fluid, from which the hay bacillus was grown. The Widal test made with these contents was negative. After the drainage of the gall-bladder the patient made a satisfactory recovery.

DISCUSSION

Altogether we have collected 18 cases of typhoid fever in children under the age of 15 who either died from, or were operated upon for, complications arising in the gall-bladder. In Case 11 of this series the acute cholecystitis did not develop until eight months after recovery from the disease; in all the other cases the complications came on during the course of the disease. Eight patients died without the interference of a surgeon. All of these cases were reported prior to the year

1893. Since that time 10 cases which have been treated surgically have been reported. One of these patients died—a mortality of 10 per cent.

The records of the earliest cases are not supported by any bacteriological studies. In recent years cultures of the gall-bladder have usually been made at the time of operation. In this connection the leucocyte count is of interest. When the cultures have shown a pure culture of the typhoid bacillus the count has been relatively low, usually about 10,000. In Case 12, in which cultures were not made, there was a leucocyte count of 33,000 and in Case 15, which showed an organism not definitely identified, there was a count of 21,000.

Thomas, in 1907, collected from the literature 154 cases of typhoid fever complicated by cholecystitis. Perforation of the gall-bladder occurred in 39 of these. Twenty-eight of the patients were not operated upon and died; 11 were treated surgically with a mortality of 54.6 per cent. In 1908, Ashhurst collected 21 cases of acute cholecystitis in which an operation was performed during the course of typhoid fever. Eight of the patients recovered. Price, in 1916, collected eight other operative cases and added one of his own, this bringing the total of operations up to 30 cases. In Price's nine collected cases there was only one death and this was due to intestinal perforation two weeks after the operation on the gall-bladder. In the surgical clinic of this hospital the results have been good. The six children treated surgically and reported in this paper have all recovered. The good results in recent years are due mainly to the fact that the operations have been performed before rupture of the gall-bladder and partly also to better surgical treatment.

Emphasis should be put upon the importance of differentiating between gall-bladder complications that do and those that do not require surgical treatment. Slight pain and tenderness in the region of the gall-bladder, associated with a slight degree of spasticity of the right rectus muscle, are not so very unusual during the course of typhoid fever. The vast majority of these patients get well. Operating on typhoid fever patients for minor symptoms of cholecystitis is probably only slightly less justifiable than operating upon all patients that have pain in order to prevent intestinal perforation. Nevertheless, the low operative mortality justifies operation when there is grave doubt as to the nature of the condition of the gall-bladder. Acute suppurative typhoidal cholecystitis should receive immediate surgical treatment, for in such cases rupture of the gall-bladder may occur, and thus lessen many times the chances of recovery. We believe that the best treatment is cholecystectomy.

BIBLIOGRAPHY

1. Alexieef: Jour. Dietskaya Meditzina, 1896, No. 4; *also in* The surgical complications and sequels of typhoid fever (Keen), 1898, 268.
2. Archambault: Bull. Soc. anat. de Par., 1852, 90.
3. Ashhurst, A. P. C.: Am. J. M. Sc., Phila., 1908, CXXXV, 541.
4. Barthez, E., and Rilliet, F.: Traité des maladies des enfants, Paris, 1853, 2. éd., II, 5, 701.
5. Blachstein, A. G.: Johns Hopkins Hosp. Bull., Balt., 1891, II, 96-103.
6. Bond, J. W.: Jahrb. f. Kinderh., 1886, XXIV, 291.
7. Camac, C. N. B.: Johns Hopkins Hosp. Rep., Balt., 1900, VIII, 339.
8. Chédevergne, S.: De la fièvre typhoïde et de ses manifestations (etc.), Thèse de Paris, 1864, No. 173.
9. Chiari, H.: Prag. med. Wchnschr., 1893, XVIII, 261-263.
10. Chiari, H.: Ztschr. f. Heilk., 1894, XV, 199.
11. Deaver, H. C.: Ann. Surg., Phila., 1919, LXIX, 534-536.
12. Eisendrath, D. N.: Ann. Surg., Phila., 1917, LXVI, 557-559.
13. Erdmann, J. F.: Ann. Surg., Phila., 1903, XXXVII, 878-882.
14. Gilbert, A., and Girode, J.: Compt. rend. Soc. de biol., Par., 1890, 9. s., II, 739-742.
15. Hagenmüller, P.: De la cholécystite dans la fièvre typhoïde. Thèse de Paris, 1876, 50 pp., 4°, No. 269.
16. Hölscher, A.: Münch. med. Wchnschr., 1891, XXXVIII, 43: 62.
17. Husson: Bull. Soc. anat. de Par., 1835, 104.
18. Keen, W. W.: The surgical complications and sequels of typhoid fever, Phila., 1898, W. B. Saunders, 386 p., 8°.
19. von Khautz, A.: Centralbl. f. d. Grenzgeb. d. Med. u. Chir., Jena, 1913, XVI, 545-548.
20. Mason, A. L.: Tr. Ass. Am. Physicians, 1897, XII, 23; *also in* Boston M. & S. J., 1897, CXXXVI, 449.
21. Mitchell, J. F.: Johns Hopkins Hosp. Bull., Balt., 1902, X, 399.
22. Nichols, H. J.: J. Exper. med., N. Y., 1917, XX, 573.
23. Osler, W.: Tr. Ass. Am. Physicians, 1897, XII, 378.
24. Pratt, J. H.: Am. J. M. Sc., Phila., 1916, CXXII, 584.
25. Price, J. W.: Kent. M. J., Bowling Green, 1916, XIV, 45-49.
26. Prince, E. M.: J. Am. M. Ass., Chicago, 1909, LII, 1581.
27. Prince, E. M.: Surg., Gyn. & Obst., Chicago, 1910, XI, 416.
28. Quénu, E.: Rev. de chir., Par., 1911, XLIII, 145.
29. Stedman, C. E.: Med. & Surg. Rep. Bost. City Hosp., 1882, 3. s.
30. Thomas, B. A.: N. York M. J. (etc.), 1907, LXXXVI, 688.
31. Webb-Johnson, A. E.: Surgical aspects of typhoid and paratyphoid fevers, 1919.
32. Welch, W. H.: Johns Hopkins Hosp. Bull., Balt., 1891, II, 121.
33. Wharton, H. R.: Tr. Am. Surg. Ass., Phila., 1909, XXVII, 540.

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X-RAY STUDIES OF THE SEMINAL VESICLES AND VASA DEFERENTIA AFTER URETHROSCOPIC INJECTION OF THE EJACULATORY DUCTS WITH THORIUM

A NEW DIAGNOSTIC METHOD

By Hugh H. Young and Charles A. Waters

(From The James Buchanan Brady Urological Institute, The Johns Hopkins Hospital)

The vast canal system extending upward from the orifices of the ejaculatory ducts in the verumontanum presents a wide field for study hitherto neglected both in urology and roentgenology. Only during the past few years has proper attention been paid to the rôle of the seminal vesicles in the production of any one of the numerous types of arthritis, cardiac and gastro-intestinal disturbances and neuroses, and up to the present time the assistance to be derived from the X-ray has been practically disregarded.

Belfield, Mills, Thomas and Pancoast and Picker have published excellent roentgenograms showing the vesicles and vasa injected with opaque substances through a vasotomy.

In the present paper we desire to call attention to a method by which the vesicles can be injected through the catheterized ejaculatory ducts following endoscopy, and, further, to emphasize the fact that, so far as our experience goes, there is no harmful effect associated with this procedure, and lastly to show that by the injection of thorium it is possible to outline the vast canal system above the orifices of the ejaculatory ducts.

It is generally stated in most text-books that strictures of the ejaculatory ducts do not occur and, as far as we can find in the literature, Luys is the only one who has attempted to explore the interior of the ducts by "catheterism."

For the past four years one of us (Young) has been endeavoring by means of specially devised probes, filiforms, bougies of metal and whalebone, and with Geraghty's utricle syringe (Fig. 1) to explore and treat the interior of the ejaculatory ducts, the vasa deferentia and seminal vesicles and in these experiments it has been found that the ejaculatory ducts are easy to locate in most cases even when not visible. A special probe will usually pass upward for a distance of 2 to 4 cm. into the vas deferens. In this way many differences in caliber have been discovered and several cases of marked stricture of the ejaculatory ducts have been made out.

In these cases systematic dilatations, done at weekly or bi-weekly intervals, have sometimes brought about almost immediate relief of chronic pain and discomfort in that region. In another paper a description of these cases, the methods employed and the results obtained will be given in detail. In the course of this work we have been struck with the need of a method by which the condition of the canal system above the verumontanum can be graphically depicted without resorting to opening the vas deferens in the groin as has been done by Belfield, Thomas and Pancoast, Mills and others.

With the introduction of thorium in making roentgenograms, it occurred to us to use this agent for the purpose of getting the much desired pictures of the inferior of the vasa deferentia and the seminal vesicles. By using Geraghty's utricle syringe, it has been found easy in most cases to inject 1 or 2 c. c. of thorium solution and to obtain excellent radiographs of the vasa deferentia out to the external rings (Figs. 2) as will be described later on.

By means of the Young urological X-ray table (Figs. 3, 4, 5, 6) it is possible to make stereoscopic X-ray plates on the urological table immediately after injecting the ejaculatory ducts as shown in the illustration.

The utricle syringe is so constructed that the slender tip, which is about 1.5 cm. long, enters the duct for that distance and is then arrested by the shoulder.

This distance is apparently sufficient to carry it always into the vas deferens, as our radiographs have never shown the seminal vesicles when this instrument was used. With a shorter cannula, injections pass into the seminal vesicles.

In the progress of our work, having found it desirable to inject both ejaculatory ducts simultaneously, we devised a special forked cannula, one branch being 3 mm. longer than the other (Fig. 1-B). With this arrangement of the terminal tubes it is easy to introduce the longer tube into one duct for a distance of about 3 mm. and then be free to catheterize the other duct with the shorter tube, after which the instrument is pushed home until arrested by the junction point of the two tubes against the anterior surface of the verumontanum. With this forked cannula we have found it possible to inject 3 c. c. of thorium solution at one time and to inject both seminal vesicles.

We have thus demonstrated that catheterization of the ejaculatory ducts and radiographic study of the canal system above may be carried out with ease and that it furnishes a ready and satisfactory method of determining the condition of these structures. The process is apparently without danger. In some 50 cases in which the instruments and thorium solution have been introduced for varying distances into the vasa deferentia and seminal vesicles, we have never encountered an epididymitis or any other deleterious sequela.

The development of this method has been the direct outcome of the combined X-ray and urological table (Young's) by means of which it is possible to take radiographs while the patient is in a position for cystoscopy without disturbing him. This table makes it possible to place the patient in the horizontal, vertical, inverted and any intermediate position and to take radiographs in any of these positions without disturbing the patient or the urologist (note different positions of table in Figs. 3, 4, 5, 6). The roentgenologist is able to change his

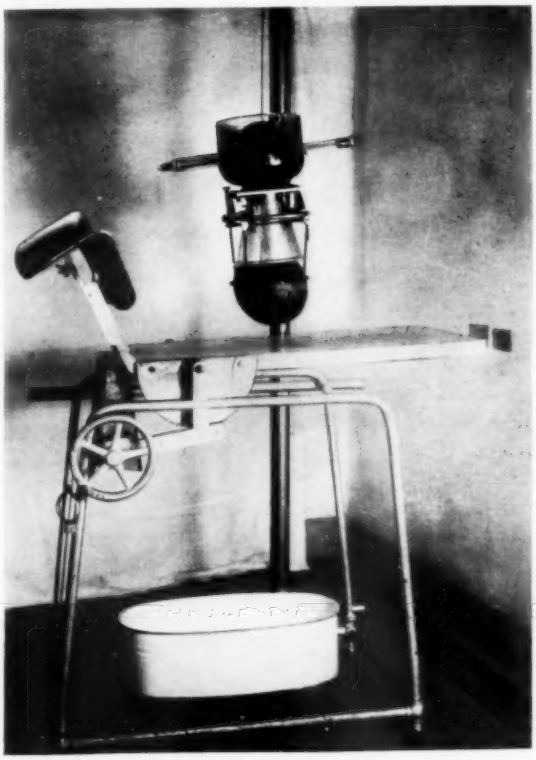

FIG. 3.—Table horizontal with tube and compression attachment in position.

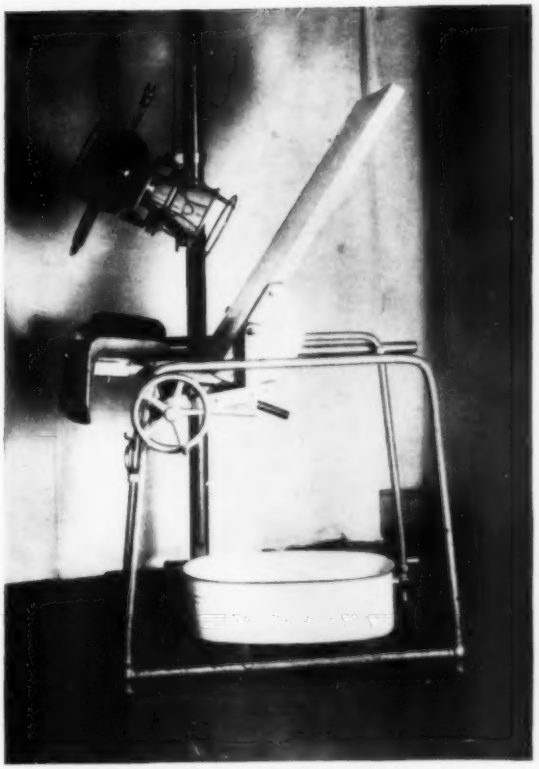

FIG. 4.—Side view of table in upright position.

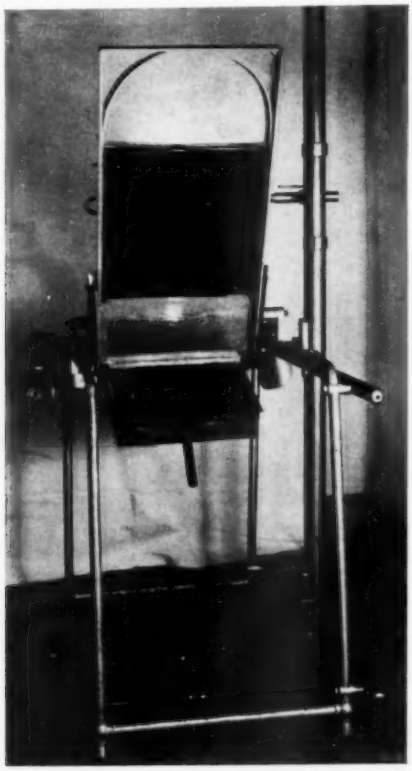

FIG. 5.—Rear view of table in upright position, showing plate carriage in position for exposure.

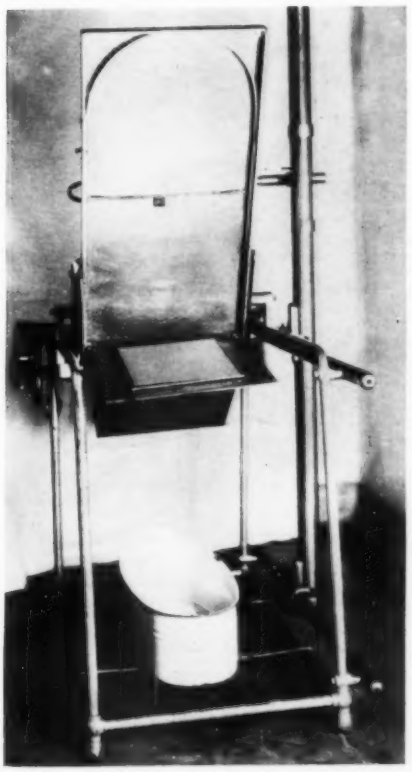

FIG. 6.—Rear view of table in upright position. Plate carriage and holder are lowered for reception of the plate.

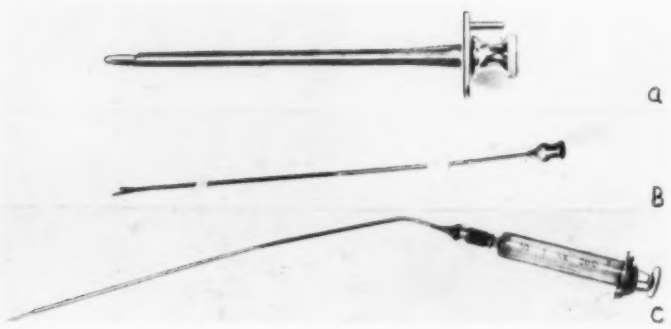

FIG. 1.—Essential instrument used in the catheterization of the ejaculatory ducts. Note the specially devised forked cannula, (B), used in injecting both ducts at the same time. One arm of the fork is 3 mm. longer than the other. A.—Endoscope. B.—Double Forked Cannula. C.—Geraghty's' Utricle Syringe.

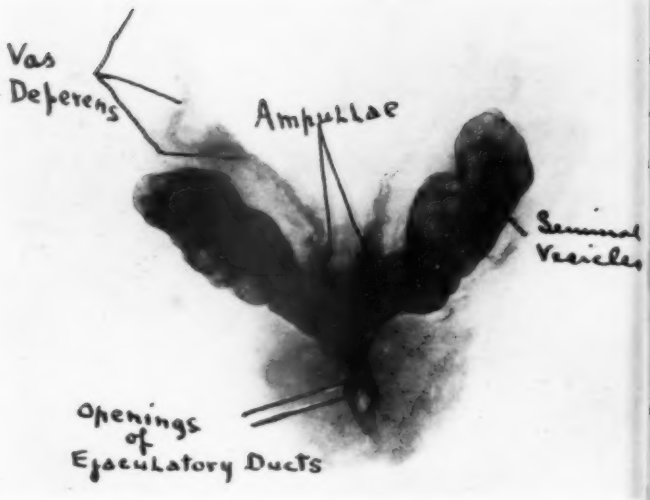

FIG. 7.—Showing the anatomical arrangement from an X-ray point of view.

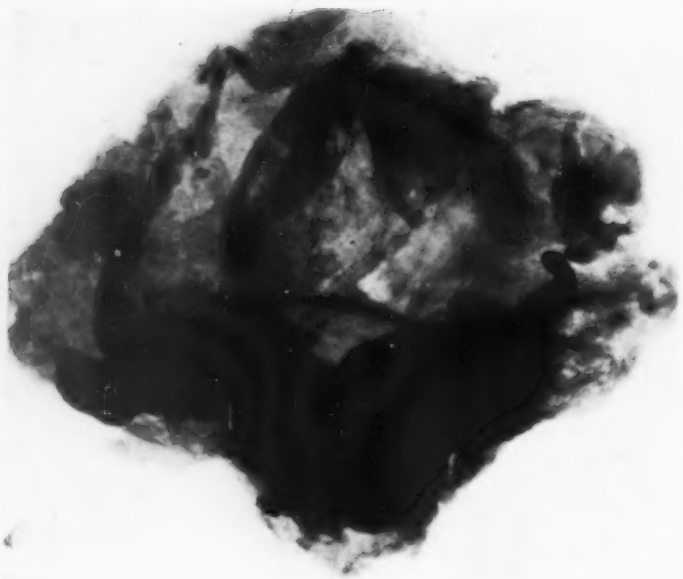

FIG. 8.—Diseased vesicles and vas, showing marked irregularity and distortion.

FIG. 13.—Drawing made from stereoscopic plates showing both vasa injected.

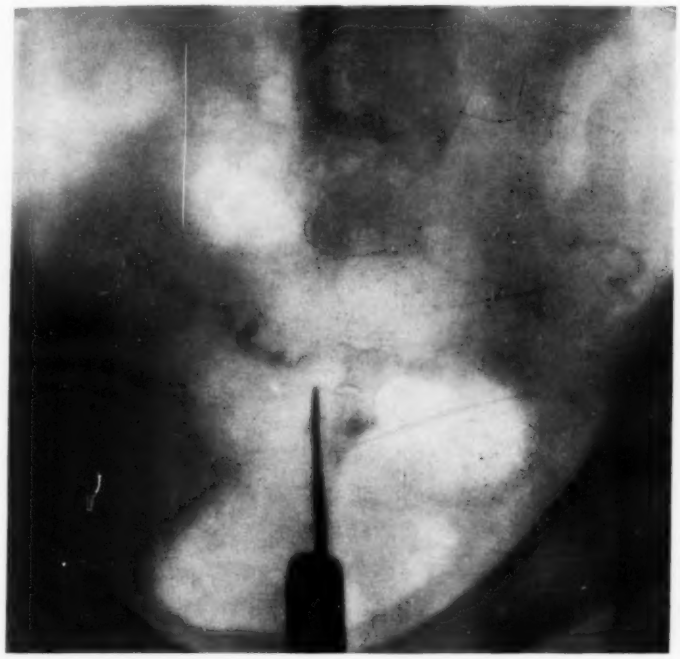

FIG. 2.—After the injection of the left side. Both vasa deferentia are now visible. The injections can be traced to the approximate location of the internal abdominal ring.

FIG. 9.—Shows the left seminal vesicle partially filled, the convolutions and sacculations appearing normal.

FIG. 10.—Injection with thorium showing the course, size and convolutions of the right vas deferens. The vesicles and left vas do not show in this plate. The syringe is seen in the lumen of the right ejaculatory duct. Left not yet injected.

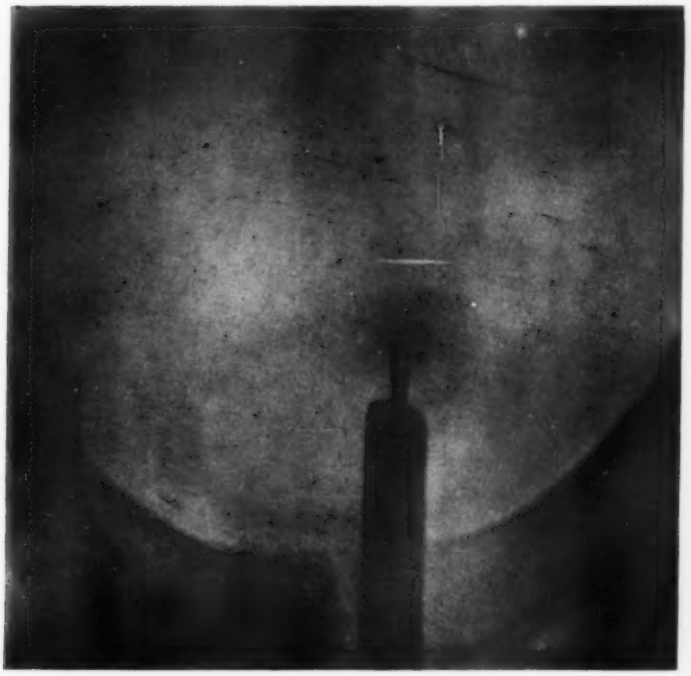

FIG. 11.—Shows the forked syringe in the lumina of the ejaculatory ducts before injection was made.

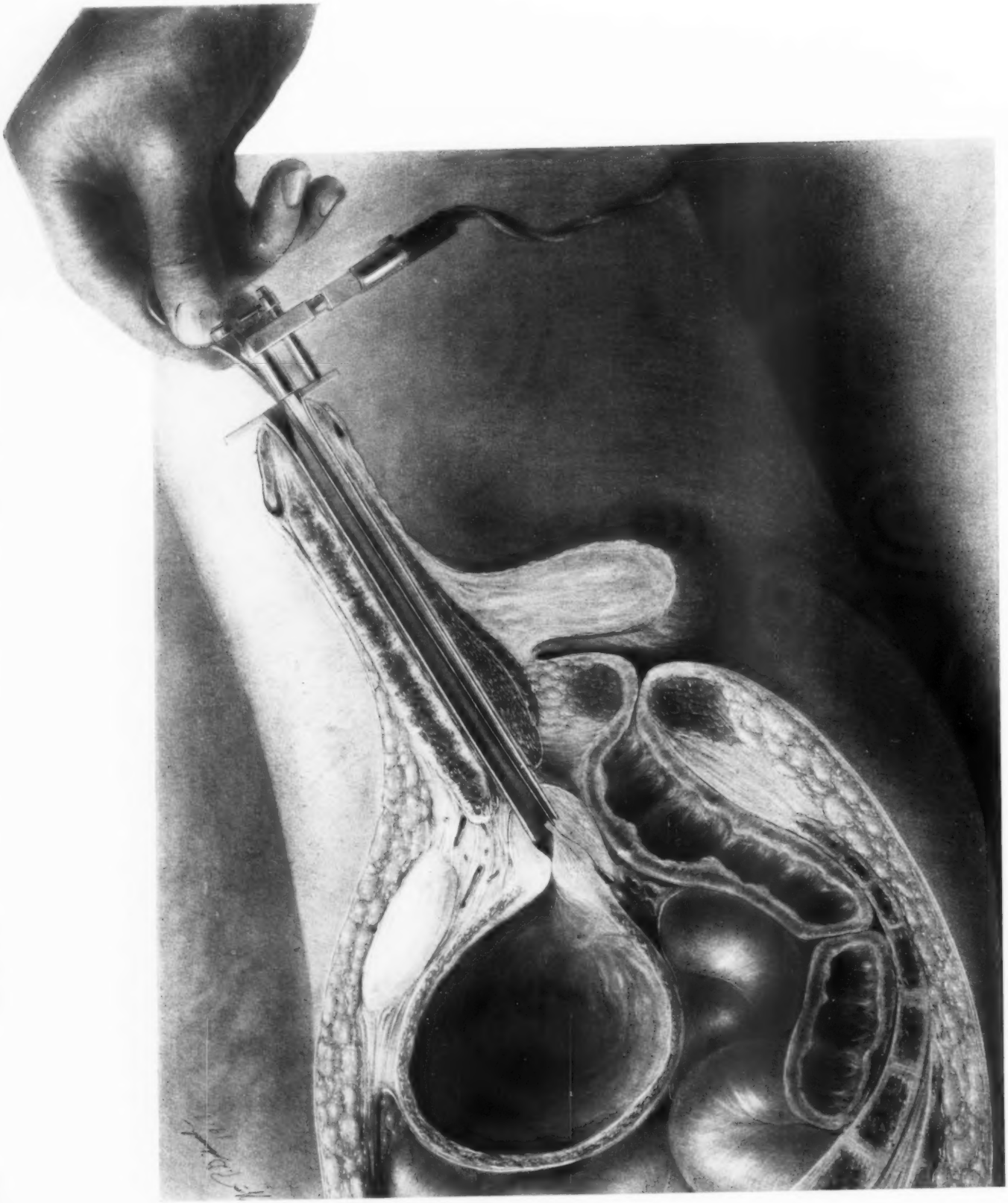

FIG. 12.—Young's method of dilating ejaculatory ducts. Probe seen entering ejaculatory duct.

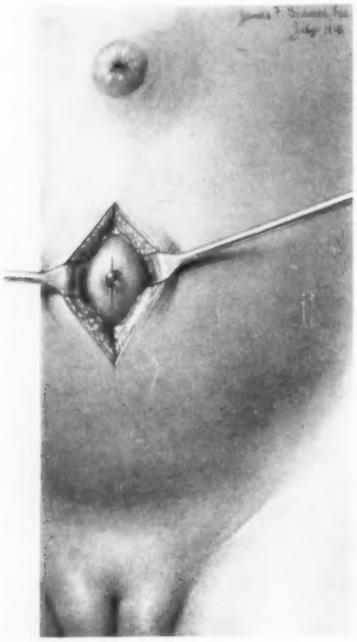

FIG. 1.—Vesicle projected through a prick-hole in the peritoneum.

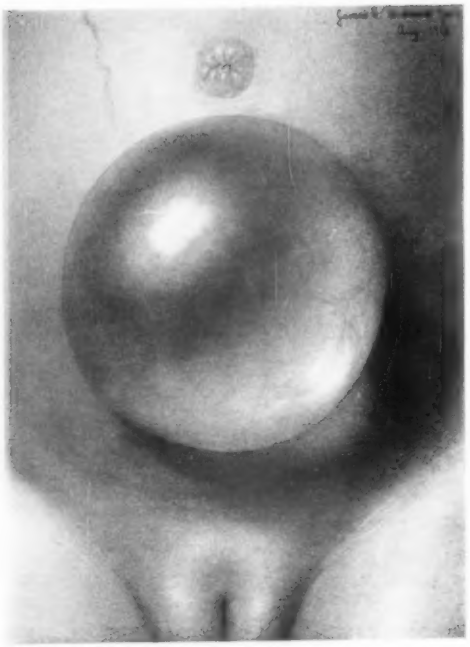

FIG. 2.—A subsequent stage of the vesicle.

FIG. 3.—Its ultimate aspect.

plates for stereoscopy under the table with ease and dispatch, as shown in Figs. 5 and 6.

From an X-ray viewpoint, the anatomical structures of interest in the making of vesiculograms are (1) the verumontanum with the external openings of the ejaculatory ducts; (2) the ejaculatory ducts themselves; (3) the seminal vesicles; (4) the ampullæ of the vasa deferentia, and (5) the vasa deferentia above the ampullæ (Fig. 7).

The following vesiculograms have been prepared from autopsy specimens with a view of showing the variations existing in the anatomical structures. The specimens have been injected with thorium through the openings of the ejaculatory ducts. The anatomy is clearly shown; the lumina of the ejaculatory ducts are plainly reproduced; while the convolutions and windings of the seminal vesicles and the ampullæ of the vasa deferentia are clear and distinct. Variations in the vesicles and vasa deferentia have been observed. One typical plate is shown here (Fig. 8). The vesicles are greatly enlarged; their borders are irregular and indistinct and there is a loss of the normal sacculations and convolutions; both ampullæ are markedly enlarged and irregular. The vasa above the ampullæ are also seen to be dilated. The whole represents the pathological picture of an obstructive inflammatory process in the vesicles and ejaculatory ducts.

In Figs. 2 and 10 the syringe is inserted straight, first on the left side and then on the right. Only the vasa are seen. In Fig. 9, the syringe is inserted at an angle and only the vesicle is seen. The explanation is that in the first instance the syringe passed by the opening of the vesicle and that, therefore, only the vas was injected, whereas, in the second instance, the syringe entered the vesicle and blocked the vas so that only the vesicle was injected.

Among the conditions for which this method seems applicable and helpful for diagnosis and treatment, the following may be mentioned:

1. To determine the patency of the ejaculatory duct or vas in cases of sterility when epididymo-vasotomy is contemplated.
2. To determine whether stricture of the ejaculatory duct, of the vas or of the outlet of the seminal vesicle is present.
3. To disclose the condition of the ampullæ of vasa or seminal vesicles in inflammatory or tuberculous conditions.
4. To show the condition of the seminal tract in studies to determine the cause of vague pain in the region of the prostate, vesicles or bladder. Other uses of this method will be forthcoming.

BIBLIOGRAPHY

1. Fuller: J. A. M. A., Nov. 30, 1912, p. 1961.
2. Picker: XIV Internat. Med. Cong., Lond., 1913; 1911. Read before the Third Congress of the German Urologic Society.
3. Luys: Traité de cystoscopie et d'uréthroscopie. Paris, 1914, Octave Doin et Fils, Editeurs.
4. Belfield: Surg., Gyn. and Obst., XVI, 1913, 569.
5. Thomas and Pancoast: Ann. of Surg., LX, 1914, 313.
6. Milstone, H. J.: Seminal Vesiculography, Wisconsin M. J., 1918, XVII, No. I, 146.

SELF-EVENTRATION OF A LARGE ABDOMINAL HYGROMA THROUGH A SCALPEL PRICK OF THE PERITONEUM

By W. S. HALSTED

On the 19th of May, 1893, a child two years old, was brought from the Out-Patient Department of The Johns Hopkins Hospital to the operating room with the request that she be tapped for ascites. On two or three occasions fluid had been withdrawn by the dispensary physicians from the distended abdomen, but on this morning repeated attempts to relieve the distension with the trochar and cannula had been unsuccessful, hardly a drop of fluid being obtained. The child's abdomen presented, as it seemed to us, the typical ascitic picture.

In making a short incision in the mid-line I accidentally pricked the peritoneum. Immediately there protruded through the prickhole a vesicle hardly larger than a mustard seed. The little bladder, slowly increasing in size (Figs. 1 and 2), soon covered the abdomen, and finally in saddlebag fashion fell over the child's flanks, a broad, flat isthmus of sac-contained fluid stretching across the now scaphoid belly from one great bag of water to the other, both of these resting on the bed-sheets (Fig. 3). The wall of this great cyst was of filmy thinness.

The mid-line incision was then lengthened and a search made for the pedicle about which several small cysts were found to be grouped. All of these seemed to have their origin in the great omentum—embryologically, in the posterior mesogastrium. An independent cyst, about as large as an orange, seemed to be contained between the layers of the duodenal mesentery, the continuation of the stomach's mesentery or posterior mesogastrium. This cyst was so adherent to the mesenteric vessels that we feared its removal might imperil the circulation of the bowel; hence we stitched its wall to the parietal peritoneum, and in a few days opened and drained it. The child made a prompt recovery.*

Twenty-two years later, in 1915, after prolonged search, this patient was finally traced and persuaded to come to the hospital for examination. She was then 24 years of age, married and in good health. Her mother stated that she had suffered no ill effects from the operation. Examination of the abdomen revealed nothing abnormal except perhaps a little tenderness in the region of the appendix.

I have found no record of a case of abdominal hygroma observed so many years after operation for its removal.

The surgeon should bear in mind the possibility of lymphatic cyst whenever a child with distended abdomen comes under observation.

* Full details of this case are recorded in the hospital history, Surg. No. 2245.

In April, 1916, an infant, aged seven weeks, was operated upon in extremity at The Johns Hopkins Hospital † for the relief of what was supposed to be intestinal obstruction. The cause of the distension proved to be a lymphatic cyst, and the child promptly succumbed to the operation. It is conceivable that this infant might have been saved, had the correct diagnosis been made and the cyst evacuated or possibly eventrated through a small incision.

† Surg. No. 39451.

THE UPTURNED EDGE OF THE LIVER OVER ACUTELY DISTENDED, EMPYEMATOUS GALL-BLADDERS

A DIAGNOSTIC SIGN OF SOME VALUE

By W. S. Halsted

A drawing made at the operating table about 20 years ago by the late August Horn depicts quite well the upturned edge of the liver over an acutely inflamed and distended gall-bladder (Fig. 1). This manifestation undoubtedly has been frequently observed by surgeons, and I direct attention to it merely on account of its occasional value as a confirmatory diagnostic sign. It may be much more pronounced than in the figured instance, and this ridge can be palpated if the abdominal wall is not too tense or abnormally thick. I cannot recall having noted this upturned or everted edge in the absence of signs of infection of the gall-bladder, but am not sure that it may not occur with hydrops vesicæ.

More than once this sign has enabled me in a debatable case to make the correct diagnosis. In one patient there was a non-resilient mass, larger perhaps than a man's fist, situated in the epigastrium and extending only slightly more to the right than to the left of the mid-abdominal line. Surmounting this mass was a linear, almost vertical ridge hardly 2 cm. to the right of the linea alba. The physicians of The Johns Hopkins Hospital who consulted me were inclined to believe that we had a new growth under our fingers, and I was of the same opinion until on defining this ridge the correct interpretation occurred to me. At operation the mass proved to be made up of large intestine, duodenum, gall-bladder and a drawn out lobe of liver matted together and infiltrated with inflammatory exudate. The lobe of Riedel had been tugged to the right and so rotated that its upturned edge occupied rather a longitudinal than a transverse position.

In quite a number of cases in which the liver's edge over the gall-bladder, not upturned, was free and pliable, I have been able to feel it through the abdominal wall and to raise it on the back of a finger-nail insinuated between the gall-bladder and this hepatic lip.

I make it a point in every case of suspected enlargement of the gall-bladder to test for an overlying free border of the liver, upturned or not, and have much more frequently made it out in the flat than in the everted state.

There can hardly be anything new to experienced operators in what I have said, but for physicians the drawing may have interest.

THE FATE OF BACTERIA INTRODUCED INTO THE UPPER AIR PASSAGES

II. *B. COLI* AND *STAPHYLOCOCCUS ALBUS*

By Arthur L. Bloomfield

(*From the Biological Division of the Medical Clinic, The Johns Hopkins University and Hospital*)

In studying the pathogenesis of infectious disease it is necessary to consider a set of factors quite apart from the inherent "virulence" or "invasive power" of the virus and the "resistance" of the tissues of the host in the stricter sense—namely, the conditions that determine the fate of the organism from the time when it reaches the body until it is eliminated or until actual invasion takes place followed, perhaps, by the production of disease. Such conditions are obviously complex and variable, and to a certain extent accidental. Thus, potentially pathogenic bacteria may reach the mouth only to be ejected or swallowed before disease can be initiated, or a small dose of organisms may perish under relatively unfavorable conditions where a part of a larger number might survive and invade the host; or anatomical variations in the polymorphous architecture of the body orifices may lead in one case to the harmless sidetracking of a germ which in another individual may perchance be deposited on a vulnerable point. On the other hand, there exist protective mechanisms more constant and orderly in their action which tend to destroy or to eliminate certain bacteria. The bactericidal effects of the mouth secretions may be mentioned as an example.

Despite the importance of exact information about the fate of pathogenic organisms during the period between their meeting with the host and their elimination or the inception of

FIG. 1.—Edge of Liver Everted by an Acutely Distended Gall-Bladder.

disease, we have at present only the limited and incomplete facts gleaned from epidemiological study. In the case of measles, for instance, it is probable that in the non-immune person inoculation is promptly and invariably followed by the production of disease—in other words, conditions are such that no effective opposition is offered to the invasion of the virus. In pneumonia, on the other hand, a different state of affairs exists. Although pneumococci are probably almost always transferred from the patient to the mouths of those in attendance, acquisition of the disease by direct contact is very unusual; and when a carrier state develops it is, as a rule, temporary.[2] It is clear, therefore, that a certain amount of opposition is offered to the free development and persistence of the pneumococcus in the mouth, and that this must be a factor in limiting the spread of the disease despite the potential virulence of the organisms and the susceptibility of the host. Similar reasoning may be applied to infections by other organisms such as the meningococcus, the streptococcus and others.

In a previous paper [1] it was suggested that information about the fate of bacteria in the upper respiratory tract might be obtained by actually placing the organisms on a definite site such as the tongue, tonsil, or nasal mucosa, and studying by repeated culturing their subsequent disposal. Sarcina lutea, which is non-parasitic and non-pathogenic for human beings, was employed. It was found that this organism swabbed in large amounts on the tongue or nasal septum or introduced into the tonsillar crypts could no longer be recovered after a brief interval, usually from one to two hours. This rapid disappearance seemed to be due to a direct destructive effect of the mouth secretions which kill the organism *in vitro* within one hour. The present paper deals with a similar study of bacteria which are usually non-pathogenic, but which are parasitic in human beings and may at times produce disease—*B. coli* and *Staph. albus*.

EXPERIMENTS WITH B. COLI

Methods.—The methods employed were essentially like those described in the previous work. The subjects were free from any marked abnormality of the upper respiratory passages and were not suffering from any acute disease. The bacteria were placed with a platinum loop on the selected site—the anterior half of the tongue, the mucosa of the nasal septum just posterior to the vestibule, or a tonsillar crypt. In the case of the last deep pocket-like crypts were chosen wherever possible so that the organisms could be deposited well within them. It required a considerable amount of practice to carry out this procedure successfully. The condition of the site of inoculation was always carefully noted and trauma was avoided. In no case was any local lesion or general reaction observed following inoculation. The patients were kept under direct observation for the first two hours of the experiment, and except in a few instances no food or fluid was taken until after the two-hour culture was made. The strain of *B. coli* used had been recently isolated from a stool and gave the typical reactions of this organism. Twenty-four hour growths on plain agar were used. The cultures were made on plain agar to which from 5 to 10 per cent of sodium taurocholate was added. On this medium practically all of the usual throat flora were inhibited and the detection of *B. coli* was easy.

EXPT. I.—*B. coli* was swabbed on the tongue and cultures were made from the tongue and the pharynx after 10 minutes, 2 hours, 24 hours, and 48 hours. The results are summarized in Table I.

Summary.—*B. coli* swabbed on the tongue was rapidly diffused over the whole mouth cavity. In three cases the organisms could no longer be recovered after 24 hours. In one instance a few colonies were recovered after 24 hours.

EXPT. II.—*B. coli* was swabbed on the mucosa of the nasal septum and cultures were made from the nose and naso-pharynx after 10 minutes, 2 hours, 24 hours, and 48 hours. The results are summarized in Table II.

Summary.—*B. coli* swabbed on the nasal septum could not be recovered from the nose after 24 hours in 3 cases. In one case a single colony was recovered after this interval. The organisms were carried rapidly to the pharynx where they were recovered in variable numbers up to 24 hours.

EXPT. III.—*B. coli* was introduced into tonsil crypts and cultures were made at various intervals for three or four days from the crypt and the pharynx. The results are summarized in Table III.

Summary.—*B. coli* introduced into tonsil crypts could be recovered after 24 hours in every case, and in one instance after as long as 48 hours. The organisms apparently were gradually discharged from the crypt over a period of from one to two days and no permanent carrier state was set up. Their persistence for a longer time in the tonsil crypts than in the pharynx (Expt. I) suggests that they are mechanically pocketed and prevented from being washed away. As long as the bacteria remained in the crypt a few could be recovered from the adjacent areas of the pharynx. In one instance (Mo) the control culture yielded four colonies of *B. coli*. This was the only control culture in the whole series in which *B. coli* was found. It probably represented a transient carrier state.

EXPERIMENTS WITH STAPHYLOCOCCUS ALBUS

Methods.—The same methods were employed as in the previous experiments, but the cultures were made on plain agar. The strain was obtained from the skin, and gave the typical cultural reactions.

EXPT. I.—*Staph. albus* was swabbed on the tongue and cultures were made from the tongue and pharynx after various intervals. The results are summarized in Table IV.

Summary.—A large dose of *Staph. albus* swabbed on the tongue was rapidly spread about the mouth cavity. Practically all the organisms disappeared within 24 hours. After this interval a few colonies were recovered in one case, but not more than in the controls.

EXPT. II.—*Staph. albus* was swabbed on the nasal septum and cultures were made from the nose and naso-pharynx after various intervals. The results are summarized in Table V.

Summary.—The majority of a large dose of organisms swabbed on the nasal septum disappeared in 24 hours. A few colonies could be recovered later but not more than were present in control cultures.

EXPT. III.—*Staph. albus* was introduced into tonsil crypts and cultures were made from the crypt and pharynx at various intervals. The results are summarized in Table VI.

Summary.—The organisms disappeared rapidly. After 24 hours one colony was recovered from a crypt in one of three cases. There was no tendency toward the production of a carrier state.

TABLE I.—FATE OF B. COLI SWABBED ON TONGUE

Date	Name	Diagnosis	Age	Procedure	Number of Colonies of B. coli per Plate Recovered From Tongue and Pharynx								Control culture before inoculation	
					After 10 minutes		After 2 hours		After 24 hours		After 48 hours			
					Tongue	Pharynx	Tongue	Pharynx	Tongue	Pharynx	Tongue	Pharynx	Tongue	Pharynx
Sept. 22.	Sp.	Tuberculous peritonitis.	16	½ slant B. coli swabbed on tongue.	∞* B. coli.	∞ B. coli.	∞ B. coli.	∞ B. coli.	No growth.	No growth.	No growth.	No growth.	No growth.	No growth.
Sept. 23.	Car.	Psychosis.	40	½ slant B. coli swabbed on tongue.	∞ B. coli.	∞ B. coli.	About 100 cols. B. coli.	About 200 cols. B. coli.	14 cols. B. coli.	1 col. B. coli.	No growth.	No growth.	A few cols. staphylococcus.	No growth.
Oct. 8.	Wa.	Myocardial insufficiency.	60	½ slant B. coli swabbed on tongue.	∞ B. coli.	About equal parts of B. coli and B. mucosus.	About equal parts of B. coli and B. mucosus.	Mostly B. mucosus. A good many cols. B. coli.	No growth.	B. mucosus.	B. mucosus.	B. mucosus.	B. mucosus.	B. mucosus.
Oct. 9	L.	Myocardial insufficiency.	55	½ slant B. coli swabbed on tongue.	∞ B. coli.	∞ B. coli.	∞ B. coli.	∞ B. coli.	No growth.	No growth.	No growth.	No growth.	No growth.	No growth.

TABLE II.—FATE OF B. COLI SWABBED ON NASAL MUCOSA

Date	Name	Diagnosis	Age	Procedure	Number of Colonies of B. coli per Plate Recovered From Nose and Nasopharynx								Control culture before inoculation	
					After 10 minutes		After 2 hours		After 24 hours		After 48 hours			
					Nose	Nasoph.	Nose	Nasoph.	Nose	Nasoph.	Nose	Nasoph.	Nose	Nasoph.
Sept. 23.	Lew.	Pleurisy.	30	½ slant B. coli swabbed on left nasal septum.	∞ B. coli.	4 cols. B. coli.	∞ B. coli.	No B. coli.	No B. coli.	2 cols. B. coli.	No B. coli.	No B. coli.	No B. coli.	No B. coli.
Sept. 23.	Lan.	Myocardial.	60	½ slant B. coli swabbed on left nasal septum.	∞ B. coli.	No B. coli.	∞ B. coli.	No B. coli.	No B. coli.	No B. coli.	No B. coli.	No B. coli.	No B. coli.	No B. coli.
Oct. 8.	Lan.	Myocardial.	60	½ slant B. coli swabbed on left nasal septum.	∞ B. coli.	No B. coli.	1000 cols. B. coli.	1200 cols. B. coli.	1 col. B. coli.	No B. coli.	No B. coli.	No B. coli.	No B. coli.	No B. coli.
Oct. 8.	Sp.	Tuberculous peritonitis.	20	½ slant B. coli swabbed on left nasal septum.	∞ B. coli.	No B. coli.	∞ B. coli.	A good many cols. B. coli.	No B. coli.	No B. coli.	No B. coli.	No B. coli.	No B. coli.	No B. coli.

TABLE III.—FATE OF B. COLI INTRODUCED INTO TONSILLAR CRYPTS

Date	Name	Diagnosis	Age	Procedure	Number of Colonies of B. coli per Plate Recovered From Tonsil Crypt and Pharynx										Control culture before inoculation	
					After 10 minutes		After 2 hours		After 24 hours		After 2 days		After 3 days			
					Crypt	Pharynx	Crypt	Pharynx	Crypt	Pharynx	Crypt	Pharynx	Crypt	Pharynx	Crypt	Pharynx
Sept. 25.	Lew.	Pleurisy.	30	One loop of B. coli placed in a tonsillar crypt.	∞ B. coli.	∞ B. coli.	8 cols. B. coli.	200 cols. B. coli.	10 cols. B. coli.	1 col. B. coli.	No B. coli.	No B. coli.	No B. coli.	No B. coli.	No B. coli.	No B. coli.
Oct. 7.	Mo.	Myocardial.	65	One loop of B. coli placed in a tonsillar crypt.	∞ B. coli.	6000 cols. B. coli.	∞ B. coli.	1000 cols. B. coli.	1000 cols. B. coli.	10 cols. B. coli.	1 col. B. coli.	No B. coli.	No B. coli.	No B. coli.	4 cols. B. coli.	No B. coli.
Oct. 7.	H.	Chronic endocarditis.	40	One loop of B. coli placed in a tonsillar crypt.	500 cols. B. coli.	No B. coli, plate overgrown by B. mucosus.	15 cols. B. coli.	No B. coli, plate overgrown by B. mucosus.	300 cols. B. coli.	No B. coli; B. mucosus.	No B. coli.	No B. coli; B. mucosus.	....	....	No B. coli.	No B. coli; B. mucosus.

* ∞ = innumerable

TABLE IV.—FATE OF STAPHYLOCOCCUS ALBUS SWABBED ON THE TONGUE

Date	Name	Diagnosis	Age	Procedure	Number of Colonies of Staphylococcus albus per Plate Recovered From Tongue and Pharynx												Control culture before inoculation	
					After 10 minutes		After 2 hours		After 24 hours		After 48 hours		After 72 hours		After 4 days			
					Tongue	Pharynx	Tongue	Pharynx	Tongue	Pharynx	Tongue	Pharynx	Tongue	Pharynx	ongue	Pharynx	Tongue	Pharynx
Oct. 14.	Fa.	Tabes.	45	½ slant of Staph. albus swabbed on tongue.	∞ Almost pure.	∞ Almost pure.	∞ Almost pure.	About 50 cols. Staph. albus.	About 50 cols. Staph. albus.	2 cols. Staph. albus.	About 10 cols. Staph. albus.	0	0	0	....	....	0	About 10 cols. Staph. albus.
Oct. 14.	Ba.	Cirrhosis of liver.	40	½ slant of Staph. albus swabbed on tongue.	∞ (pure).	∞ (pure).	∞ Almost pure.	10 cols. Staph. albus.	1 col. Staph. albus.	0	....	....	....	....	....	....	1 col. Staph. albus.	0
Oct. 14.	Ca.	Hypertension.	46	½ slant of Staph. albus swabbed on tongue.	∞ (pure).	∞ Almost pure.	∞ Almost pure.	20 cols. Staph. albus.	0 Staph. albus.	0 Staph. albus.	....	....	....	....	....	....	0	0

TABLE V.—FATE OF STAPHYLOCOCCUS ALBUS SWABBED ON NASAL MUCOSA

Date	Name	Diagnosis	Age	Procedure	Number of Colonies of Staphylococcus albus per Plate Recovered From Nose and Nasopharynx												Control culture before inoculation	
					After 10 minutes		After 2 hours		After 24 hours		After 2 days		After 3 days		After 4 days			
					Nose	Na o-pharynx	Nose	Naso-pharynx	Nose	Naso-pharynx	Nose	Naso-pharynx	Nose	Naso-pharynx	Nose	Naso-pharynx	Nose	Naso-pharynx
Oct. 15.	Wa.	Myocardial disease.	60	½ slant Staph. albus swabbed on left nasal septum.	∞ Almost pure.	0	Plate overgrown by spore bearer.	20 cols. Staph. albus.	Plate overgrown by spore-bearer.	0	20 cols. Staph. albus.	20 cols. Staph. albus.	....	....	10 cols. Staph. albus.	0	About 100 cols. Staph. albus.	0
Oct. 15.	Sp.	Peritonitis (tuberculous).	20	½ slant Staph. albus swabbed on left nasal septum.	∞ Almost pure.	0	∞ Almost pure.	20 cols. Staph. albus.	100 cols. Staph. albus.	10 cols. Staph. albus.	1 col. Staph. albus.	0	....	....	....	....	About 10 cols. Staph. albus.	0
Oct. 15.	Ma.	Convalescent pneumonia.	25	½ slant Staph. albus swabbed on left nasal septum.	∞ Almost pure.	0	∞ Almost pure.	10 cols. Staph. albus.	10 cols. Staph. albus.	0	0	0	5 cols. Staph. albus.	0	....	....	A few cols. Staph. albus.	1 col. Staph. albus.

TABLE VI.—FATE OF STAPHYLOCOCCUS ALBUS INTRODUCED INTO TONSILLAR CRYPTS

Date	Name	Diagnosis	Age	Procedure	Number of Colonies of Staphylococcus albus per Plate Recovered From Tonsil Crypt and Pharynx												Control culture before inoculation	
					After 10 minutes		After 2 hours		After 24 hours		After 2 days		After 3 days		After 4 days			
					Crypt	Pharynx	Crypt	Pharynx	Crypt	Pharynx	Crypt	Pharyux	Crypt	Pharynx	Crypt	Pharynx	Crypt	Pharynx
Oct. 16.	Ha.	Chronic endocarditis.	40	One loop of Staph. albus placed in a tonsil crypt.	∞ Pure.	0	∞ Almost pure.	0	1 colony Staph. albus.	0	0	....	....	....	....	....	0	0
Oct. 14.	Falk.	Neurasthenia.	50	One loop of Staph. albus placed in a tonsil crypt.	∞ Pure.	A good many colonies Staph. albus.	∞ Pure.	A good many colonies Staph. albus.	0	1 colony Staph. albus.	0	A good many colonies Staph. albus.	....	....	....	....	0	A good many colonies Staph. albus.
Oct. 14.	Ba.	Myocardial insufficiency.	30	One loop of Staph. albus placed in a tonsil crypt.	∞ Pure.	∞ Mixed plate.	∞ Pure.	Many colonies Staphylococcus albus.	100 colonies Staph. albus.	0	1 colony Staph. albus.	3 colonies Staph. albus.	....	....	....	....	0	0

EXPLANATION OF THE DISAPPEARANCE OF *B. COLI* AND *STAPH. ALBUS*

As in the previous study with sarcina lutea an attempt was made to investigate certain factors which might be responsible for the disposal of the organisms, to wit, the reaction of the mouth secretions, mechanical removal by normal flushing processes in the mouth and nose, the destructive action of mouth secretions, and the possible antagonistic action of other bacteria present in the mouth.

The Antagonistic Action of Mouth Bacteria.—The question of the antagonistic action against invading organisms of bacteria present in the mouth opens a broad field of study about which we have at present very little exact information. Our impression is that the bacteria already present played no part in the disposal of *B. coli* and *Staph. albus.* This impression is based on the fact that these organisms throve in mixed culture with the mouth bacteria in almost every case. In one individual (Ha), who was a carrier of *B. mucosus,* the plates were regularly overgrown by this organism and it may have played a part in preventing other bacteria from gaining a foothold.

Reaction of Mouth Secretions.—No practicable method was available for determining accurately the reaction of special regions of the mouth or nose. Furthermore, this did not seem advisable, inasmuch as the constant flux of secretions must lead to a continual alteration of the hydrogen-ion concentration. Systematic studies on this point now in progress have already shown that the pH of the mouth secretions may vary considerably from day to day in the same individual. A mixture of expectorated saliva and mouth secretions was therefore collected and its reaction was regarded as corresponding in general to that of the mouth surfaces. The pH of such secretions tested by the colorimetric indicator method has varied in about 50 observations made under various conditions from 6.2 to 7.4. Both *B. coli* and *Staph. albus* grew apparently with equal luxuriance on media whose reaction covered this range. Clark,[3] also, found the final hydrogen-ion concentration of cultures of *B. coli* on various media to be below pH 5.0. It seems unlikely, therefore, that the reaction of the mouth in itself explains the disappearance of these bacteria, although variations in pH may play a part in preventing or favoring rapid growth.

The Bactericidal Effect of the Mouth Secretions.—Equal amounts of a freshly expectorated mixture of saliva and mouth secretions were added to suspensions of a 24-hour growth of *B. coli* and *Staph. albus.* A standard loop of the mixture was plated at various intervals, and smears were made at the same time. An illustrative protocol of one of several experiments which gave essentially the same result follows (Table VII).

From this experiment it may be concluded that neither *B. coli* nor *Staph. albus* is killed by the mouth secretions in 24 hours. The experiment may be criticized in that the number of organisms used was very large and that a smaller number might have been inhibited or destroyed. It was desired, however, to determine only any gross destructive action which the mouth secretions might exercise. The whole subject of the influence of salivas of various composition and reaction on the initiation of bacterial growth and the latent period requires further study.

Mechanical Influences.—After it had been determined that the mouth secretions exercised no destructive effect on *B. coli* or *Staph. albus,* other explanations for the disappearance of these bacteria were sought. The question immediately arose whether simple mechanical removal by the normal flushing of the mouth and nose by secretions or ingested fluid could account for their disappearance. It seemed desirable, therefore, to study the fate of inert particles placed in the mouth, nose and tonsil in a way similar to that employed in the case of the bacteria. For this purpose *Kieselguhr* and *rotten earth* were used. The former is a diatomaceous earth which is readily recognized under the microscope, the latter consists of insoluble crystals of characteristic form about the size of a red blood cell or larger, also easily recognizable. One or two loops of a thick saline paste of these substances was placed on the tongue, on the nasal septum, and in tonsil crypts. Scrapings were made at various intervals and examined microscopically. The

TABLE VII.—EFFECT OF MOUTH SECRETIONS AND SALIVA ON B. COLI AND STAPHYLOCOCCUS ALBUS

B. COLI

One standard loop from each tube plated at following intervals	Tube 1 0.2 c. c. mouth secretions pH 7.2	Tube 2 0.2 c.c. suspension B. coli	Tube 3 0.2 c.c. suspension B. coli + 0.2 c. c. mouth secretions	Tube 4 0.05 c. c. suspension B. coli + 0.2 c. c. mouth secretions
Immediately.	No growth. (Bile agar).	B. coli.	B. coli.	B. coli.
2 hours.	No growth. (Bile agar).	B. coli.	B. coli.	B. coli.
24 hours.	No growth. (Bile agar).	B. coli.	B. coli.	B. coli.

STAPH. ALBUS

One standard loop from each tube plated at following intervals	Tube 1 0.2 c. c. mouth secretions pH 7.0	Tube 2 0.2 c.c. suspension Staph. albus	Tube 3 0.2 c.c. suspension Staph. albus + 0.2 c. c. mouth secretions	Tube 4 0.05 c. c. suspension Staph. albus + 0.2 c. c. mouth secretions
Immediately.	Abundant colonies of various kinds.	Staph. albus.	Staph. albus, almost pure.	Staph. albus, almost pure.
2 hours.	Abundant colonies of various kinds.	Staph. albus.	Staph. albus, almost pure.	Staph. albus, almost pure.
24 hours.	Abundant colonies of various kinds.	Staph. albus.	Staph. albus, almost pure.	Staph. albus, almost pure.

results of these experiments were uniform and striking. They may be summarized as follows:

TABLE VIII

FATE OF INERT PARTICLES (KIESELGUHR AND ROTTEN EARTH) PLACED IN THE UPPER AIR PASSAGES.

Kieselguhr placed on the tongue could not be detected after 2 hours.
Rotten earth placed on the tongue could not be detected after 2 hours.

Kieselguhr placed on the nasal septum was detected after 2 hours but not after 24 hours.
Rotten earth placed on the nasal septum was detected after 2 hours but not after 24 hours.

Kieselguhr placed in a tonsil crypt was detected after 24 hours but not after 48 hours.
Rotten earth placed in a tonsil crypt was detected after 24 hours but not after 48 hours.

These experiments show that inert particles placed on the tongue, nasal septum, or in the tonsil crypts disappear at about the same rate of speed as *B. coli* or *Staph. albus*. The rapid disappearance from the nose at first glance seems especially remarkable, but it is in no way incompatible with what is known about the rate at which particles are transported by ciliated epithelium. Furthermore, as soon as the particles reach the inferior meatus the lachrymal secretion rapidly washes them to the posterior nares.

It appears, then, that while no direct destructive action is exercised upon *B. coli* or *Staph. albus* by the mouth secretions either *in vivo* or *in vitro*, conditions are such that the bacteria are removed by mechanical processes more rapidly than they multiply unless protected in some spot such as a tonsil crypt, where they remain relatively stagnant. There seems to be no tendency for them to take hold and grow diffusely in the mouth or nose.

DISCUSSION

The present report represents part of a systematic study of the fate of bacteria introduced into the upper air passages. This study has so far revealed two distinct mechanisms for the disposal of such organisms. The first illustrated by sarcina lutea consists of a prompt and direct destruction of the bacteria by the mouth secretions. The second, illustrated by *B. coli* and *Staph. albus*, seems to apply to a group of organisms which have no tendency to grow diffusely or to localize in any part of the upper air passages, but which on the other hand are not directly destroyed by any bactericidal action. These bacteria seem to be removed by the normal mechanical flushing processes in the mouth and nose. It is possible that the study of other organisms will reveal still other mechanisms for removing invading bacteria.

The fate of *Staph. albus* brings up points of particular interest. This organism was selected for study because it may be recovered at almost any time in small numbers from the nose or mouth of normal individuals. We wished to determine whether the introduction of a massive dose would lead to a permanent carrier state or whether the organisms would disappear rapidly. It was found that they disappear at about the same rate as *B. coli*, the number of bacteria recoverable promptly falling to the average or normal level. This observation seems to us to indicate that the few staphylococci ordinarily found in the mouth or nose are simply transients introduced from without the body, and that they are not actually growing or multiplying there as parasites. This presumption suggests further the general principle that some other members of the so-called mouth flora are organisms which are in transit into and out of the mouth and that they are not actual parasites. The whole subject of the constant variations of the mouth flora and their significance will be discussed at another time.

Observations such as those here reported raise the complex question of the relative favorableness and unfavorableness of the upper air passages as sites for bacterial growth. It should be emphasized that in dealing with environment we must consider every variation from conditions which are positively destructive to those which are simply unfavorable in the sense that a certain degree of resistance is offered to the initiation of optimal bacterial growth. An analysis of such conditions must include a consideration of certain qualities of the bacteria themselves such as the stage of growth, the size of the inoculum and the nature of the organism (source, virulence, etc.), as well as a consideration of the qualities of the medium, such as its reaction, certain chemical elements in its composition, the presence of other organisms, etc. These conditions are obviously endlessly complex and to a large extent defy exact analysis. It has seemed advisable in the present experiments, therefore, simply to gather the general facts about the disposal of organisms hoping later to deal more intelligently with particular details.

CONCLUSIONS

1. *B. coli* and *Staph. albus* swabbed on the tongue or nasal septum usually disappeared within 24 hours.
2. *B. coli* and *Staph. albus* introduced into tonsil crypts could be recovered after somewhat longer intervals.
3. In no case was a permanent carrier state set up.
4. Inert particles disappeared at about the same rate of speed as the bacteria.
5. The organisms probably disappear because they are mechanically removed more rapidly than they multiply.
6. The disposal of *B. coli* and *Staph. albus* illustrates a mechanism radically different from that effective in removing sarcina lutea.

REFERENCES

1. Stillman, E. G.: J. Exp. Med., 1916, XXIV, 651.
2. Bloomfield, A.: Bull. Johns Hopkins Hosp., 1919, XXX, 317.
3. Clark, W. M.: Jour. Biological Chemistry, 1915, XXII, 87.

A CASE OF NON-PARASITIC HÆMATOCHYLURIA

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At the present time there are probably not more than a dozen cases of non-parasitic chyluria to be found in the literature. Several patients whose cases have been diagnosed as non-parasitic have lived in localities where filariasis is common and the diagnosis has rested upon the failure to demonstrate the embryos in the blood. Great caution should be used in diagnosing as non-parasitic the case of a patient who has dwelt in a filarial district, because the majority of chyluria cases result from infection by filaria sanguinis hominis. Filarial cases are known to clear up and the embryos disappear from the blood, so that we should reserve the term non-parasitic for the cases of those who have never been exposed to infection from filaria. Filariasis is an uncommon condition in the United States, although occasionally met with in Southern ports.

The case which I wish to report occurred in a young woman, 23 years of age, who has always lived in the mountains of Virginia:

Miss C. S., aged 23, from the mountains of Virginia, came in our clinic August 5, 1919, on account of the remarkable appearance of her urine and general weakness. The family history was essentially negative. The patient had never been strong and vigorous since an attack of chorea at nine years of age. Since that time she had always remained delicate, but had been able to assist in the household duties of her home. She had been rather nervous and for several years a facial tic had bothered her. At rather long intervals she had been subject to syncopal attacks which had caused her to lose consciousness upon several occasions. She usually had the attack after exercise and was always conscious of their approach. She had never bitten her tongue or had incontinence of urine during any of these attacks.

She had never had any pulmonary symptoms and her appetite had always been good. For a period of nine years, or since she was 14 years of age, she and her family had noticed an unusually milky appearance of her urine, which was always more marked in the morning. The urine at times had been perfectly clear, but for nine years, since the condition was first noticed, there had been no period of more than a few days that it had remained so. During the greater part of the time the patient had not been conscious of having any bladder or kidney trouble outside of the remarkable appearance of the urine. She had never been troubled with retention or incontinence, but she had frequently noticed that her stream would be interrupted by the passing of a clot. She could not recall ever having had attacks of pain that would suggest renal colic, and fever had never accompanied any of her bladder symptoms. She had noticed more frequent voiding when exercising but she had never been troubled with nycturia.

Her menstrual history was perfectly normal. The patient had weighed about 100 pounds for the past few years.

The following are the important physical findings:

The *thyroid gland* is slightly enlarged but there are no symptoms of thyroid intoxication.

Lungs.—Essentially normal.

Heart.—Mitral stenosis and insufficiency (compensated).

Abdomen.—No tenderness or rigidity. Liver and spleen not enlarged. No tenderness on deep pressure over the gall-bladder region or McBurney's point. No abdominal masses made out. Kidneys in good position, not enlarged.

Pelvis.—Virginal outlet; rectal examination negative.

August 7: Cystoscopy by Dr. H. H. Hampton. No. 8 Kelly cystoscope passed.

Bladder normal in appearance. Ureteral orifices hard to find; normal in appearance. Right ureter catheterized with No. 8 renal wax-tipped catheter. No obstruction to catheter. A specimen from the right kidney urine, which is fairly clear, shows few red blood cells. Bladder urine, milky; contains red blood cells, leucocytes and oil droplets.

August 7: Functional test: Right kidney—collected through renal catheter; left kidney, from the bladder.

Amount of urine	Phenolsulphonphthalein excreted
Right kidney 1 hour 15 c. c.	18%
Left kidney 1 hour 20 c. c.	15%
	33%

The urine from the right kidney was clear. The bladder urine was slightly milky.

August 11: Cystoscopy. A No. 8 Kelly cystoscope passed and a No. 8 bismuth renal wax-tipped catheter passed. Specimen from the kidney showed a clear urine with red blood cells. Culture taken. X-ray taken before and after a thorium injection shows a fairly large kidney shadow with the calices incompletely filled. Capacity normal, ureter not dilated, no stone or other shadows.

August 14: Cystoscopy. A No. 8.5 cystoscope passed after a bladder culture had been taken. Bladder urine milky, shows many red cells and a few leucocytes. The bladder wall was examined carefuly for ulcers or diverticula, but none were found. The bladder was everywhere normal in appearance. The left ureter was catheterized but no urine was obtained. When fluid was injected it produced pain in the left flank. On palpation through the rectum, a thickened ureter could be felt on the left; the right ureter was not palpable. [This induration of the ureter might come from the passage of the catheter or tuberculosis.]

August 29: Cystoscopy. The bladder urine is milky in appearance, the cystoscopy having been done about two hours after lunch. The bladder is perfectly normal in appearance. There is a silght reddening of the ureteral orifices due to former catheterization, more marked upon the left side. Both ureters were catheterized with No. 8 wax-tipped renal catheters. Specimens of urine from both sides contained red cells and a few leucocytes; neither specimen was milky. There were no fat globules. No bacteria in hanging drop. No ova seen. Culture taken.

The patient was returned to the ward and given a meal—the regular ward diet, a fair amount of butter and two glasses of milk. Urine collected separately from both kidneys every hour for four hours showed the following:

First hour: Right kidney, very milky, but less so every hour until almost clear the last hour. None of the left kidney specimens was milky, but all contained traces of blood.

September 2: Cystoscopy. A No. 8 Kelly cystoscope passed. Bladder normal in appearance. A No. 8 bismuth catheter passed two-thirds of the way up to the right kidney. Culture taken. Pelvis capacity about 15 c. c. An X-ray taken before and after injection. Good picture obtained showed slightly dilated pelvis. Large kidney shadow. Ureter not dilated. Very small tortuous

linear shadows leading off from tip of calices into renal cortex. These shadows were rather indistinct and could be seen only in the negative.

Altogether the patient was subjected to five cystoscopic examinations within a period of four weeks. Cultures were taken from the kidneys and bladder at each sitting and all were reported negative except one bladder culture which was evidently a contamination (Staphylococcus albus). The bladder and kidney specimens were carefully examined a dozen or more times for tubercle bacilli but none were ever found. The bladder was given a careful routine examination at each sitting before catheterization of the ureter and was always found normal in appearance. Nothing resembling the mouth of a sinus was seen. Milky urine was always obtained from the bladder and large quantities of water were necessary to free the bladder of oil, even when the urine from the kidney was clear. I might mention here that it is not uncommon to find oil droplets in the urine that has remained in the bladder a week or more, following previous treatment, so that milky urine from the bladder is not positive evidence that the ureter is execreting milky urine at that particular time. For this reason both ureters should be catheterized simultaneously in investigating chyluria cases.

Several differential blood counts were made, all of which showed a slight eosinophilia. Otherwise there was nothing remarkable. The red count and hæmoglobin were higher than one would expect in a patient suffering from a constant loss of blood; the red count being 6,123,000 and the hæmoglobin 90 per cent. The fresh blood and smears were repeatedly examined for filarial embryos, but none were ever found. Wassermann test negative.

Blood Study.—On admission R. B. C. 6,123,000. W. B. C. 9160. Hgb. 90 per cent.

Differential Count	P. M. N.....	75%
	P. M. B.....	0
	P. M. E.....	6%
	Lymph.	14%
	Trans.	3%
	Smudges	2%
		100%

The X-ray of the thorax showed nothing suggestive of pulmonary tuberculosis.

Urine.—The urine varied in character throughout the day, being most remarkable in appearance early in the morning, and almost normal at times during the day. The first specimen voided in the morning (when the patient was on a fat-containing diet) was always milky and, upon standing, a light red, jelly-like clot would form and float near the top of the vessel, a bloody sediment collecting on the bottom. Portions of the clot were examined repeatedly, but nothing but blood cells and fibrin were ever found. The urine did not have the normal urinous odor, especially when fresh, but had rather a fleshy smell. Freshly voided specimens were usually acid, but some of the early morning specimens were alkaline. All specimens contained red blood cells in abundance, many leucocytes and albumin. No sugar was ever found. No parasites or ova were ever seen. The specific gravity varied from 1006 to 1018, the early morning specimen usually showing the highest for the day. No casts were ever found, all the specimens examined contained red blood cells and leucocytes.

Experiments With Diet.—Simple experiments with the diet were made over a five-day period, the urine being collected in four-hour portions. These were examined separately chemically and microscopically. Upon the first day the patient was given ordinary ward diet, which contains a moderate amount of fat. All the four-hour specimens were milky except the midnight specimen, which was cloudy. All specimens contained red blood cells, leucocytes and albumin. There was never any sugar. Altogether 1565 c. c. were voided. After the individual tests had been made upon the four-hour specimens, the remaining urine was collected in a single bottle and a fat determination made upon an aliquot portion with the Babcock lipometer, such as is used in rapid milk analysis. This gave 1.4 per cent fat, or 21.9 grams for 24 hours. Upon the second day the same diet was used, but water was forced. The patient excreted 2505 c. c. of urine with a fat determination of 1.2 per cent for the 24 hours, which gave a loss of 30 grams of fat. Each of the four-hour specimens was milky. The specific gravity was highest in the early morning, being 1012; lowest at noon, 1005. All specimens showed red blood cells, leucocytes, albumin and fat droplets; no sugar.

On the third day the patient took water as desired, but no food. The 4 a. m., 8 a. m. and 12 noon specimens showed fat in decreasing amounts. The 4 p. m. and 8 p. m. and subsequent specimens were fat-free. The 24-hour urine amounted to 1240 c. c. with an average of 0.2 per cent fat or 2.4 grams. It is perfectly evident that, had an interval been allowed after food, before the starvation specimens were saved, there would have been no fat in the urine upon the starvation day. On the following, or fourth day, the patient was given a fat-free diet with water as desired. The total urine amounted to 1750 c. c. All specimens were fat-free, which is ample proof that the urine would have been fat-free under starvation had time been given for the lymph to free itself of fat.

Upon the fifth and last day, the patient was given a fat diet with water as desired. All the four-hour specimens were milky. They contained an average of 1.6 per cent and a total of 2290 c. c. of urine was excreted, representing 36.6 grams of fat., roughly, about 30 per cent of the fat ingested, the fat intake being about 100 grams.

These experiments with the diet were not intended for a study of the fat metabolism, but were designed to prove that our patient was suffering from a true chyluria. They are very instructive, however, and would have been more so if we had determined the fat intake accurately and made fat determinations upon each four-hour specimen.

A second experiment was tried to shed some light upon the effect of posture upon the fat content of the urine.

Upon two successive mornings she was requested to empty her bladder and was given two glasses of milk before breakfast. One morning she was kept in bed and the next morning she was allowed to walk around the ward. The urine was collected every half hour for two hours. Upon both mornings all four specimens were slightly milky—the urine excreted while the patient was in bed containing a greater amount of fat than the specimens voided while she was on her feet.

We were anxious to conduct other experiments upon our patient but she wanted to return home and, as there was nothing to be done for her in a therapeutic way, we did not feel justified in holding her longer.

The literature upon this subject is not very large, although it extends over a period of half a century. Carter,[1] in 1862, presented two very interesting cases; one in an adult Hindoo, who had lymph scrotum and chyluria; the other in a Parsee youth who had a mass in his groin with a discharging lymph sinus. These cases of lymph scrotum that occur frequently in filariasis and this case of lymph sinus in the groin show the mechanical nature of the condition. All of these cases demonstrate that the rupture in the lymphatic system is due to mechanical blockage whether it be associated with inflammation or not.

Waters,[2] in 1862, reported a case of chyluria from which he drew the following conclusion: "I have come to the conclu-

sion that the affection is one in which the main pathological feature is a reflex condition of the capillaries of the kidneys, that as a consequence of this condition, the albumin, the fibrin, the fat and the blood corpuscles are filtered from the blood vessels and make their appearance in the urine." In Waters's case the condition cleared up (as often happens with filarial patients); he felt that good results had been obtained from the administration of gallic acid by the mouth.

Lüdke [3] in 1908, collected 10 cases of non-parasitic chyluria from the literature and reported a case of his own, in a woman 46 years of age. Hemoglobin, 90%; R. B. C., 4,408,000; W. B. C., 12,800. Urine acid, seldom neutral. She excreted from 6 to 8 grams of fat to 1000 c. c. of urine, which is a slightly lower excretion than in our case. The fat disappeared from the urine on fasting. He felt that his case was due to a connection between the lymphatic system and bladder, which he attributes to a colon cystitis.

Magnus Levy,[4] in a splendid article (1908), reviews the literature and reports a case of chyluria associated with diabetes in a man 51 years old. The condition was of six years' duration and unilateral, the chylous urine coming from the right kidney. The percentage of fat in the urine varied from 0 to 1.8%—about the limits in our case.

D. W. Carter,[5] in 1916, made a study of the fat content of the urine in a case of chyluria probably of filarial origin. He made repeated examinations daily for over a month upon his case. With a diet of 66.39 grams of fat, there was an average daily output of 6.45 grams. The amount of fat in the urine did not increase in direct proportion to the amount of fat ingested.

Stuertz,[6] in 1903, reported a case of chyluria as due to the presence of Eustrongylus Gigas in the left kidney, but since the chyluria did not clear up after nephrectomy, it seems very doubtful whether the two conditions were in any way related.

After reviewing a number of these cases, one cannot help being impressed by the influence of posture upon this condition.

Hnatek [7] reported a case of bilateral chyluria in a patient 33 years of age, who put out twice as much fat in the urine when in bed as when up and about; and four times as much fat from the left kidney as from the right.

Charteris [8] reports a case in which the chyluria was practically controlled by posture. The patient was an Austrian, 59 years of age, who had visited Egypt, India and Brazil. So far as he knew, he had never been infected with filaria. His morning urine was always milky but cleared up completely whenever he was upon his feet. Chyluria could be produced at any time by drinking a glass of milk and reclining at an angle of more than 45 degrees. The chyluria was most pronounced when the patient lay flat upon his back. Charteris concluded that "the chyluria was due to some intermittent pressure upon the thoracic duct by some factor that only became operative when the patient assumed certain attitudes." The patient would not submit to cystoscopy.

In our own case the morning specimen always contained the greatest amount of fat, although our experiment to demonstrate the influence of posture was not very conclusive.

The great majority of chyluria cases are periodic, our case being a notable exception.

Salkowski,[9] in 1907, reported a case of chyluria in a young man, 17 years of age, in whom the right kidney was responsible for the condition. This patient would have periods of three or four months in which his urine would be entirely normal, after which his chyluria would return. This periodical clearing up of chyluria has often led clinicians to attribute cures to many bizarre methods of treatment.

Pathological reports upon chyluria cases have been few in number. Mackenzie,[10] in 1882, reported an interesting case of filarial origin that has been frequently referred to in the literature, and I take the liberty of reproducing notes from his paper.

A soldier, 26 years of age, who had lived in India for more than a year, developed a serious chyluria, with attacks of left renal colic. Filarial embryos were demonstrated in the blood and urine. The urine contained the greatest amount of fat at night, but when the hours in bed were reversed, the day urine contained the greater amount of fat. The presence of embryos in the blood was also reversed by keeping the patient in bed during the day and letting him up at night. The patient died and following is the necropsy note:

The thoracic duct commences in a dense mass of lymphatic tissue and glands, which extends from the bifurcation of the aorta below to the level of the aortic opening in the diaphragm above. Looked at from behind, this mass occupies the whole of the space between the kidneys and is continuous below with the chains of lymphatic tissue on the iliac arteries.

The mass consists very largely of the enormously dilated lymph sinuses which can here and there be inflated. The receptaculum chyli commences by two large lymph sinuses about the size of a pencil, one from each side of the aorta and is joined opposite the aortic opening of the diaphragm by a third large sinus about the same size. The duct now ascends sinuous and much pouched for 3 to 4 inches, varying in diameter from ⅜ to ½ inch, pervious for the first one and a half inches above the aortic opening of the diaphragm, then filled with a loose clot for one and a half inches, after which it is lost in a tough thick mass (inflammatory?). The occluded point in the thoracic duct, when opened, was found to contain a very long twisted clot, tapering at the end. It is left *in situ*, to be shown, before being submitted to microscopical examination. About four inches above this point, when it can be traced, although still involved in dense tissue, it is now the size of a small crow's quill, impervious and bending to the left side behind the aorta. At its termination in the angle between the left subclavian and internal jugular veins, it passes through a mass of lymphatic tissue, is pervious and about the size of a goose quill. As stated, the iliac, the lumbar and the renal lymphatics are very much enlarged and the enlargement is specially marked in the left iliac and the left renal lymphatics. Scattered throughout the left renal lymphatics are numerous hard masses, some the size of a pea but mostly smaller. These masses manifestly occupy the lymphatic sinuses.

Mackenzie did not feel sure as to the point of communication between the lymphatic and urinary systems, but he thought that it most probably was in the varicose lymphatics of the left kidney.

Haselburg [11] described a case in which the autopsy revealed a fistulous sinus (which exuded lymph) between the bladder and a tumor that extended up to the region of the left kidney.

Manson [12] observed at autopsy that the thoracic duct was obstructed and he removed a thrombus from its lumen. The connecting fistula with the urinary tract could not be demonstrated, but he noticed dilated vessels in the cut sections of the kidneys that he took to be dilated lymphatics.

Low [13] in his case found a practically normal lymphatic system except dilated lymphatics of the kidney from which he removed a calcified filaria. Varicose lymphatics were found in the bladder wall, but no fistulous opening was demonstrated. Low felt that he was dealing with a lymphuria that came through both kidneys and bladder.

In all three of these autopsy cases there were varicose lymphatics of the kidneys; in two of them there were demonstrable obstructions in the thoracic duct. In the third case, although there was no demonstrable obstruction of the duct, dilated lymphatics in both kidneys and bladder were found. All the cases were of filarial origin.

The clinical pictures of chyluria cases are remarkably similar, whether they are of filarial origin or not, provided the patients are not suffering from an active infection. In none of the cases I have reviewed have the kidneys been reported as being markedly diseased. The patients, unless suffering from active filarial infection, are usually in fair health with very few symptoms referable to the urinary tract, except occasional discomfort from the passage of clots. In none of the cases in which the blood picture is given was there any marked secondary anæmia, although blood was usually present in the urine. Many of the patients are undernourished, but are able to live upon an ordinary diet.

The etiology of the filarial cases is understood, but in the non-parasitic it is obscure. The chyluria seems to be most severe when the patient is in bed, the early morning specimen showing the highest fat content. In all cases there is more or less blood in the urine.

Magnus Lævy [4] and Brandenburg [14] report interesting cases of chyluria associated with glycosuria.

What then is the best explanation of this condition? I believe that practically all cases depend upon mechanical obstruction of the lymphatic system, the obstruction usually being in the thoracic duct or large lymphatic trunks. The same factors are probably responsible for lymph fistulas of the lower extremities and scrotum. In most chyluria cases the chyle is found to be coming from one or both kidneys.

The cases of Haselburg [15] and Lüdke [8] demonstrate the occurrence of this lymphatic leak in the bladder, but some other bladder cases, in which the diagnosis was based upon a single cystoscopic examination without catheterization of the ureter, will not bear close analysis. We should not accept a diagnosis of bladder fistula until the kidneys have been excluded by bilateral catheterization of the ureters, the patient being put to bed and given a fatty meal and the urine collected for four hours. The first time our patient was cystoscoped her right ureter was catheterized and normal looking urine (except for a few red blood cells which are not unusual in the majority of catheterizations) was obtained. The catheter was left in one hour for a functional test and the urine remained clear during that period. It was very difficult to wash the bladder clean of fat and every cystoscopist knows how common it is to find small bladder diverticula that might be mistaken for fistulous openings.

One observer reports a case in which he bases his diagnosis upon a single bladder examination with a water cystoscope, neither ureter being catheterized. He could not see any cloudy urine being discharged into the bladder, but found a fistulous opening back of the trigone around which a scum had accumulated. The clouding around the cystoscope was so intense that he withdrew it and found some milky fluid similar to that which he had observed in the urine. The chance of error under such circumstances is so obvious as not to need discussion.

We may safely assume, then, that in most of these cases the lymphatic leak is in the kidney. The few autopsies available tend to support this statement, but the actual connection between the lymphatics and kidneys has not been identified in a single case, nor is it likely to be demonstrated until the lymphatics are injected before removal of the kidneys. I believe that the kidney lymphatics represent the point of lowest resistance in the lymphatic system and when the thoracic duct or large trunks leading from the kidneys are blocked, they rupture just as an esophageal varix does under portal obstruction, or as the cerebral arteries do in hypertension cases. This theory will explain the similarity of most chyluria cases and will also explain the fact that, as a rule, the kidney function is not interfered with. It seems evident that the deep lymphatics of the kidney are involved and that they rupture into the tubules or directly into the calices. If this fistulous connection between the systems were dependent upon inflammatory lesions of the kidney, we would probably meet with the condition oftener, and we would also find diseased kidneys more frequently associated with chyluria.

SUMMARY

In summing up our case, we find that we are dealing with a case of non-parasitic chyluria that has persisted over a period of nine years in a young woman whose health is below par. She has a mitral stenosis that has given symptoms at intervals, for more than 10 years. Her chief complaint has been general weakness with frequent syncopal attacks. Just how much of her disability has depended upon one or other of these conditions, it is difficult to say. Apparently her kidney function is unimpaired. There is no evidence of renal or pulmonary tuberculosis. From our simple experiments and clinical data we may draw the following conclusions:

(1) We are dealing with a case of non-parasitic chyluria.

(2) The leak in the lymphatic system in our case is located in the right kidney.

(3) Upon starvation or a fat-free diet, the urine becomes fat-free, blood cells and albumin persisting.

(4) Posture influences but does not control the amount of lymph leakage in our case.

(5) Increased water intake and urine output increases the "fat loss."

(6) The leakage of lymph in most chyluria cases is located in one or both kidneys and location of this connection in the ureter or bladder should be made with caution.

(7) The connection between the lymphatic and urinary systems, which is usually located in the kidneys, probably consists of ruptured varicose lymphatics belonging to the deep lymphatic system of the kidney.

(8) Lymph fistulæ, wherever they occur, are generally associated with mechanical blockage of the large trunks or thoracic duct.

I wish to thank the head of our department, Dr. Thomas Cullen, for permission to publish this case. My thanks are also due to Dr. Greenwald who did most of the clinical work.

BIBLIOGRAPHY

1. Carter, H. V.: Med.-Chir. Tr., Lond., 1862, XLV, 189-208.
2. Waters, A. T. H.: Med.-Chir. Tr., Lond., 1862, XLV, 209-223.
3. Lüdke, H.: Münch. Med. Wchnschr., 1908, LV, 1369-1373.
4. Magnus Levy, A.: Zeitschr. f. klin. Med., 1908, LXVI, 482-504.
5. Carter, D. W.: Arch. Int. Med., 1916, XVIII, 541-550.
6. Stuertz: Deutsches Arch. f. klin. Med., 1903, LXXVIII, 557-587.
7. Hnatek, J.: Deutsche Med. Wochenschrift, Leipz. u. Berl., 1910, XXXVI, 794-796.
8. Charteris, F.: Lancet, Lond., 1911, II, 1011.
9. Salkowski, E.: Berl. klin. Wchnschr., 1907, XLIV, 51-54.
10. Mackenzie, S.: Tr. Path. Soc., Lond., 1882, XXXIII, 394-410.
11. Manson: Davidson's Hygiene and Diseases of Warm Climates, 814.
12. Low: J. Lond. School Trop. M., 1911-12, I, 243-250.
13. Havelburg, W.: Arch. f. path. Anat. [etc.], 1882, LXXXIX, 365-376.
14. Brandenberg, F.: Deut. med. Wchnschr., Leipz. u. Berl., 1909, XXXV, 934-935.
15. Salle, V.: Deut. med. Wchnschr., Leipz. u. Berl., 1909, XXXV, 142-143.
16. Pope, S.: Calif. State J., San Fran., 1909, VII, 285-288.

A MYOMA OF THE UTERUS SHOWING UNUSUAL DEGENERATIVE CHANGES

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Mrs. M. G., aged 43, a patient of Dr. Amelia V. Zimmerman, entered the Church Home and Infirmary, May 6, 1919, complaining of an abdominal tumor. The family as well as the past general history of the patient was entirely negative. The menstrual history was of some interest. The onset of the menses was at 16; the duration of the periods was from three to five days and the patient had always suffered with a moderate degree of dysmenorrhœa. During the last 10 years this dysmenorrhœa had been constantly increasing until, at the time the patient entered the hospital, she was suffering with bearing down pains in her abdomen and pains in the legs for two weeks of every month. For six months previous to her admission there had been a moderate amount of yellowish vaginal discharge. There was no history of intermenstrual bleeding, or urinary symptoms, or of any loss of weight.

The general physical examination was entirely negative, the patient being a well-developed woman, with no complaints except those which were referable to her pelvic condition. On pelvic examination, the external genitalia were normal. There was no pus in the urethra, but there was a moderate amount of yellowish vaginal discharge. The outlet was marital, but not relaxed. The cervix was high in the vault, firm, pointed forward, slightly lacerated. The fundus was about twice the normal size, irregular in outline, tightly fixed in the pelvis. The adnexa could not be felt.

Pre-operative diagnosis: Myomata uteri.

On May 7, 1919, Dr. Thomas S. Cullen removed the uterus, the appendix and the right tube and ovary. Through a midline incision the pelvic organs were exposed. The uterus was found to be enlarged and to have undergone myomatous changes. On the right a large cystic mass extended out into the broad ligament and arising from the posterior surface of the uterus and extending into the left side of the abdominal cavity there was a large myoma. The operation was rendered extremely difficult by the large size of this cyst in the right broad ligament, to which the tube and ovary on that side were densely bound by adhesions and also because the right tube and ovary were bound tightly to the pelvic wall. There was considerable danger of cutting the ureter because of the extreme distortion of the pelvic organs, but by first exposing the ureter and following its course to the bladder before removing the tumor Dr. Cullen overcame this danger. The patient made an uneventful recovery and was discharged from the hospital in excellent condition three weeks after the operation.

Pathological Report.—Specimen No. 24993. The specimen consists of the right tube and ovary and the uterus with a large cyst attached. The tube is 8 cm. long. There are many adhesions around the tube, but its lumen is patent. There are no pathological changes to be seen with the microscope. The ovary measures 4 x 2 x 1.5 cm. and contains a small hæmorrhagic cyst. The microscopic picture shows a corpus luteum cyst but is otherwise negative. The uterus with the large cyst attached measures 9 x 14 x 6 cm. On opening up the uterine canal, the endometrium is seen to have a yellowish tint. Sections from several portions of the uterus show a general myomatous condition with considerable variation in the number of cells, but nowhere is there any sign of malignancy. The cyst extending out into the right broad ligament is multilocular, heart-shaped, has a bluish color and extends downward from the lower surface of the uterus. On the inner

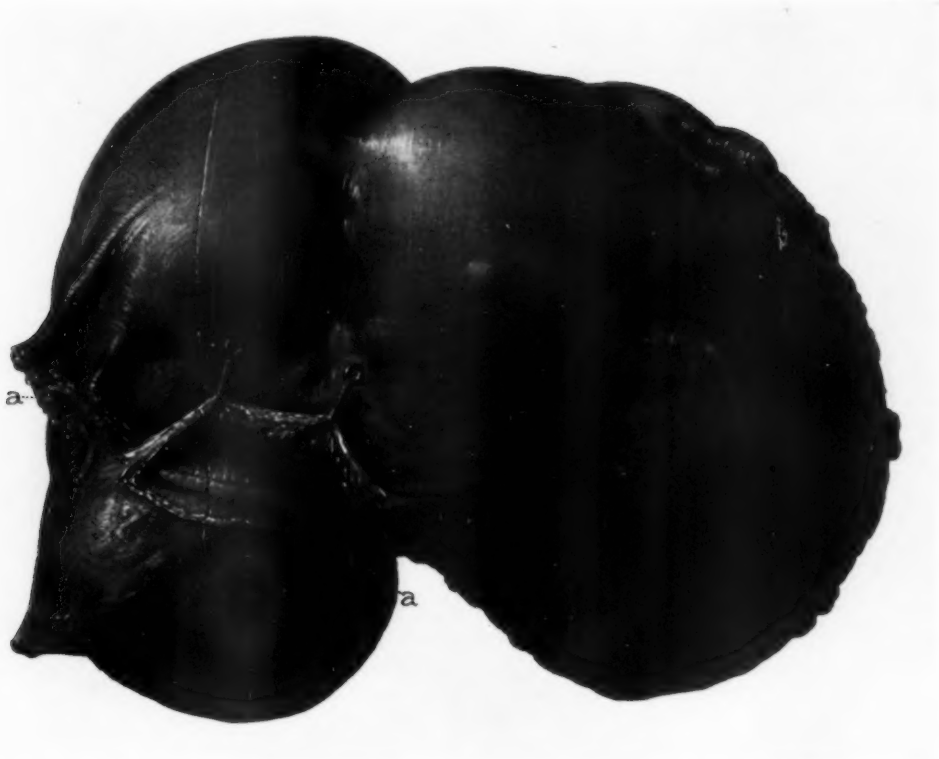

FIG. 1.—*a*, *a* shows course of duct running from the lateral wall of vagina out to the broad ligament; *b* shows line where cyst was cut.

FIG. 2.—Cross-section of cyst at *b*. *a* shows cross-section of duct. *m* is myoma on left and posterior surface of uterus.

FIG. 3.—Low-power microphotograph: On right we see marked hyaline changes with the hyaline deposited especially around the blood vessels.

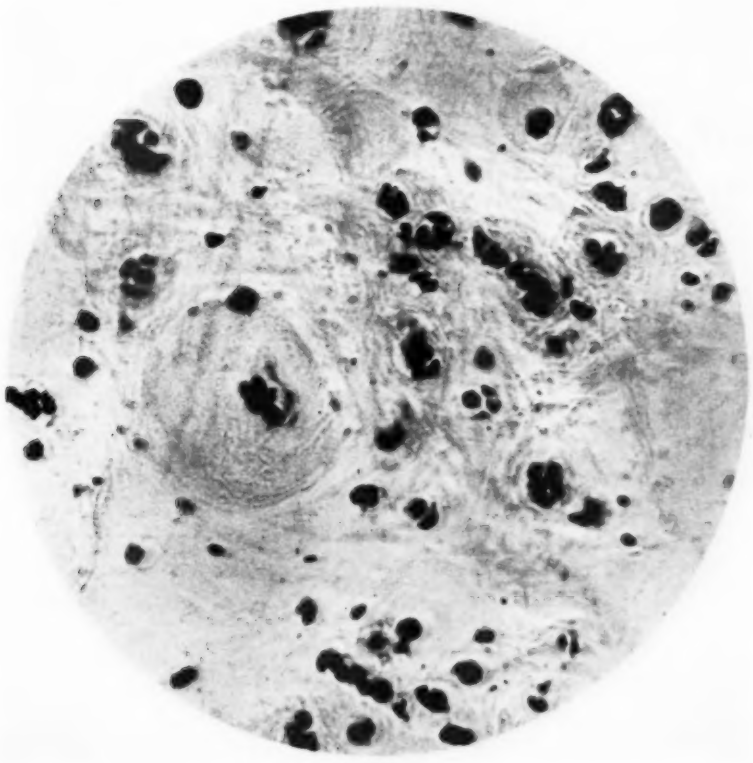

FIG. 4.—High-power microphotograph: The groupings of the nuclei in clefts resembling lacunæ is here shown.

and lower side of the cyst, about 2 cm. from the external os of the uterus, there is in the vagina an opening about 2 mm. in diameter. A small probe was introduced into this opening and a definite canal dissected out. This canal is well shown in Fig. 1, being marked by *a* and *a*.

Such a canal as here shown, extending upward from the lateral wall of the vagina along this cystic tumor, and then upward to the broad ligament, is in exactly the correct location for one of the rather rare embryonic remains of Gartner's duct. If this is the remains of Gartner's duct, the specimen is especially interesting, because we have here an opening of considerable diameter and a tube of uniform calibre, whereas in the majority of such reported cases the opening has been much smaller and the size of the lumen has varied greatly, being dilated in some portions, contracted in others. We know we are not dealing with a ureter here, because the course of the ureter was carefully traced at operation both before and after removal of the tumor and because sections of this duct show no transitional epithelium. Finally, we may have here an aberrant blood vessel anastomosing the ovarian and uterine arteries. Of course we know that ordinarily there are some anastomotic branches to these two systems, but to have a vessel of such caliber in such a location is certainly unusual. Cross-sections of the duct show a musculature resembling that found in an artery and no epithelial lining. Usually an epithelial lining is found in a Gartner's duct, but in some cases this disappears.

The cyst was opened through the anterior wall at the point *b* shown in Fig. 1. A considerable amount of fluid poured out. The cyst is made up of several smaller cysts, each of which is filled with grumous material. For the most part the material is soft and fat-like in consistency, but on passing a knife over the cut surfaces, we get here and there a gritty sensation. In one of these smaller cysts there is a well-developed polyp. Sections from the cyst show that we are dealing with a myoma showing degenerative changes. First of all we have marked hyalinization with the hyaline deposited especially around the blood vessels. In numerous areas, as in the one shown in the right-hand side of the low-power microphotograph, only the shadows of smooth muscle can be made out, while in the high-power field we see only a few deeply stained nuclei and it is hard to be sure that we are not dealing with embryonic or indeed fully developed cartilage, for the heavily stained nuclei occupying clefts resembling lacunæ remind one strongly of cartilage cells. The clefts, however, we know are caused by the contraction of the smooth muscle fibers as they undergo degeneration, as is also the grouping of the nuclei.

I wish to extend my thanks to Dr. Thos. S. Cullen, through whose courtesy I am allowed to report this case, and to Mr. Max Brödel who made the drawings of the specimen.

THE NEW WOMAN'S CLINIC AND THE CHANGE IN THE STATUS OF THE DEPARTMENTS OF GYNECOLOGY AND OBSTETRICS

On commemoration day, February 22, 1919, it was announced that an anonymous benefactor had expressed the intention of giving sufficient funds to erect upon the hospital grounds a building to serve as the woman's clinic for the medical school, and a few days later the General Education Board notified the university that beginning October 1, 1919, it was prepared to supplement the amount formerly expended by the hospital and medical school for the maintenance of the obstetrical department, so as to make it possible to place its teaching staff upon the so-called "full time" or university basis.

These generous benefactions have brought within sight the realization of long-felt hopes concerning that department, and at the beginning of the current academic year, Dr. Williams assumed his new duties, and henceforth will devote his entire time and energy to the hospital and medical school, although the department cannot be completely reorganized until the new clinic is ready for occupancy.

The donor has stipulated that "The clinic shall be devoted to the care and treatment of women before, during and after childbirth, and of women suffering from the various abnormalities of the generative organs, and shall afford provision for, and foster research into, any condition associated with the functioning of those organs."

It is hoped that the work of construction may be started early in the spring of 1920, and according to the plans now being perfected, the clinic will be an H-shaped structure occupying the sites of the present isolation, obstetrical and colored wards, and utilizing such portions of the existing buildings as may be practicable. It will consist of a basement, three full stories and a partial fourth story.

In the basement will be offices for the medical staff and for administrative purposes, teaching rooms and quarters for students on duty, a library, a museum and storage room. The first floor will be devoted to colored ward patients, both gynecological and obstetrical; the second floor to white ward patients; the third floor to private and semi-private obstetrical patients and to the research laboratories; while the fourth floor will be occupied by the gynecological and obstetrical operating rooms, the delivery rooms and the necessary space for supplies, preparation and administration. Gynecological private patients will be accommodated in the Marburg Building.

After the reorganization, which cannot be fully accomplished until the new building has been completed, the scope of the obstetrical department will be somewhat extended and will be under the direction of Dr. J. Whitridge Williams on the full-time basis.

As the result of the resignation of Dr. Howard A. Kelly, Dr. Thomas S. Cullen was made professor of clinical gynecology and placed in charge of this department, which in the future will be a sub-division of the department of general surgery.

The establishment of the woman's clinic will fill a long-felt need, and it is hoped that by placing obstetrics upon an adequately endowed full-time basis the patients will receive ideal care, more clinical material will be available for the instruction of students, and lastly, the opportunities for effective research will be greatly increased. It is also believed that in making gynecology a subdivision of general surgery a step has been taken which will prove of great benefit to students and particularly to internes and residents, in that they will have a training in the broader field of abdominal surgery rather than in a limited specialty embracing only a part of the surgery of the abdomen.

NOTES AND NEWS

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NOTES ON NEW BOOKS

Nervous and Mental Disease Monograph, Series No. 30. The Internal Secretions and the Nervous System. By DR. M. FAIGNEL-LAVASTINE, of Paris. (*New York and Washington: Nervous and Mental Disease Publishing Company, 1919.*)

This monograph is a translation by Dr. F. T. Robeson, of New York, of an address written by the author for the Congress at Berne in 1914 and published in the Revue de médecine. It is 57 pages long and as one might expect, when such a large subject is thus briefly treated, it is only a tabulation and classification of possible symptoms. As such it will prove of value for quick reference to those who are already familiar with the subject. For those who are not, the discussion is too brief to afford a comprehensive grasp of the various syndromes mentioned.

F. A. E.

Trench Fever. By MAJ. W. BYAM, R. A. M. C. and Collaborators. (*London: Henry Frowde; Hodder and Stoughton, 1919.*)

This book represents the report of investigations carried out at Hampstead for the War Office Trench Fever Investigation Committee of the British Army. It contains chapters on the symp-

tomatology of the acute and chronic stages of trench fever, on prognosis and treatment, prophylaxis, immunity, pathology and the distribution of louse-borne diseases in the world. But the greater part of the book is an account of experiments on the mode of transmission of trench fever and the conclusions to be drawn. The results of these experiments, in entire agreement with those of the American Commission, leave no doubt in one's mind that trench fever is a louse-borne disease. This point having been established, the prophylactic indications are clear, and the chapter on this subject is well written and comprehensive. Probably more than any other chapter, that on the chronic stage of the disease will be of value to the profession in civilian work. Here it is pointed out that the disease may persist for many months and the symptoms of this chronic malady are discussed. Weakness, insomnia, cardiac instability, nervousness in a discharged soldier should suggest to his physician the possibility of a chronic stage of a previous acute trench fever.

There is a good index. The book is a satisfactory summary of the current knowledge of trench fever and should be useful as such. Its value would have been enhanced, however, by more care in recording references, for even short as the literature is on this subject, this very important factor in a book of reference has been largely neglected. At the end of the book there is an abstract of the report of the American Trench Fever Commission.

F. A. E.

Psychoses of the War, Including Neurasthenia and Shell Shock. By H. C. Marr, M. D., Lt.-Col. R. A. M. C. (Temp.) Fellow of the Royal Faculty of Physicians and Surgeons, Glasgow. (*London: Henry Frowde; Hodder & Stoughton, 1919.*)

According to the preface, this book is the result of observations on 18,000 men, "the number being almost equally divided between those suffering from neurasthenia and shell shock and those affected by obvious mental disorders." The introductory chapter, containing a review of brain anatomy and physiology based on unsubstantiated theories, assumes that the silent areas are the seat of the mind; then follows a section on the observation of mental phenomena which is limited to a description of facial expression in the psychotic and feeble-minded, pathological muscular movements, and the stigmata of degeneration! In the book there is no mention of the conflict of instincts and interests, no attempt to understand the individual from the standpoint of behavior. In fact, psychogenic factors are denied. The author claims that hereditary instability or inherent weakness of the nervous system in the majority of the cases and actual shock in those with negative histories account for the dissociation of the nervous mechanism. This is meant in a literal sense, since neurasthenia is defined as weakness of the neurone.

The second chapter covers all the war neuroses material, which the author discusses under the term hereditary and constitutional psychoses, dividing them into neurasthenia (shell shock) and neurasthenia of toxic origin which includes hysteria. The work ignores completely all the modern stimulating studies of the neuroses and the following quotation illustrates the type of sterile speculation which is employed throughout the book. "Simple neurasthenia acts by reducing the tone of the bodily system generally, so that when any sudden shock such as results from the explosion of a shell causes direct injury to the body, the abdominal sympathetic system is invariably affected, and the normal resistance of the intestinal tract to organisms is broken down, and their entrance, or the entrance of their products into the circulatory system, takes place." The hysterical features may persist as habits after the so-called neurasthenia has disappeared. The author acknowledges the value of suggestion, but lays little emphasis on it in the short section on treatment which consists mainly in general measures and tonics.

The remaining chapters resemble an old-fashion text-book of psychiatry, never getting away from the explanation by inherent weakness of the neurones and auto-intoxication. The book adds nothing to the study of human behavior.

A. S.

War Neuroses and Shell Shock. By F. W. Mott, M. D., Brevet Lt.-Col. R. A. M. C. (T.) (*London: Henry Frowde; Hodder & Stoughton, 1919.*)

Dr. Mott writes in an interesting manner of his experience with the war neuroses. He has not attempted to organize the material into chapters but has presented numerous short sketches without any definite arrangement. There follows in rapid succession 124 topics with no exhaustive discussion but with many brief quotations from authorities who have undertaken more special investigations. The range of the material is great and the book is therefore lacking in coherence. The author reviews the theory of shock, distinguishing between emotional and commotional shock (commotio cerebri). The mixed type predominates and in his opinion the psychogenic factor is by far the most important cause of the phenomena following the immediate results of the explosion. He gives some cases to illustrate the acute state of mental confusion, memory disturbances, various hysterical manifestations, etc. There is a tabulation of Wolfsohn's investigation showing that war neuroses were rarely associated with wounds, that the vast majority of cases had a neuropathic soil. A few pages scattered through the book are devoted to dreams, psychoanalysis and its technique. The various psychoses including those due to alcoholism and CO gas poisoning are discussed. The question of CO gas poisoning receives more detailed attention and is accompanied by plates showing the pathological changes in the nervous tissue. Brief mention is made of the various measures used in treatment of the war neuroses. The book instead of being useful as a systematic survey is so fragmentary that it gives one only a bewildering impression of the manifold problems that confronted the neuro-psychiatrist in the war.

A. S.

Clinical Microscopy and Chemistry. By T. A. McJunkin, M. D. Cloth, $3.50. (*Philadelphia and London: W. B. Saunders Co., 1919.*)

The author has attempted in this volume to present the subject from the laboratory point of view, leaving the clinical interpretations for the instructor in Clinical Microscopy.

As a manual it serves a definite purpose though it may be said that there are a few things which might be added and some improved upon. For example, under agglutinating tests for blood to be used in transfusion, the method of using hollow ground slide preparations, which is probably the best method and in fact the one practically always used, is not mentioned.

It is true that in a brief discussion of various subjects not all the laboratory findings in various diseases can be mentioned; however, it would seem that an important finding, such as an increase in the percentage of eosinophiles in the blood of patients suffering from hookworm infection should be mentioned when outlining the clinical and laboratory findings in the disease.

The fact that an increase in the large mononuclears and transitionals so often occurs in cases of Hodgkin's disease should be mentioned under the blood findings in this disease.

For ready reference for laboratory methods this book will be useful, although there seems to be little use for more books on this subject unless they are to include new methods.

H. M. C.

Clinical Diagnosis: A Manual of Laboratory Methods. By James Campbell Todd, M. D. Cloth, $3.00. (*Philadelphia and London: W. B. Saunders Company, 1918.*)

As a manual of laboratory methods for the student or general practitioner this book apparently fulfills all requirements. The

descriptions are clear and concise. None of the commonly used laboratory methods have been omitted and some newer ones have been added. Among these are vital staining of red blood corpuscles, and the method of urobilin estimation in the stools as an aid in the diagnosis of pernicious anæmia.

There is a clear description of the method for matching bloods for transfusion purposes and also for determining easily the group to which an individual belongs. This latter method I have not found described in other text-books on clinical diagnosis.

The Wright and Kinnicutt method for counting blood platelets, which is perhaps the most simple and at the same time an accurate method, is here clearly described. Most of the books on clinical diagnosis mention this method but fail to describe it in detail, so that it becomes necessary to look up the original article to get the desired information.

The author has not intended his book for the trained laboratory worker but for the student and general practitioner, and for them it will prove very satisfactory.

H. M. C.

Chronic Traumatic Osteomyelitis. By J. RENFREW WHITE, M. D. (N. Z.), F. R. C. S. Cloth, $3.00. (*New York: Paul B. Hoeber, 1919.*)

The great war has left many men maimed from all of the belligerent nations; among them none are to be pitied more than the sufferers from chronic traumatic osteomyelitis. The author of the book under review, in his introduction deprecates the fact that no treatment proved successful in preventing the dread sequelæ to compound fractures.

"In England," he says "we have as yet seen no results in fracture cases comparable to those of Leriche, no results comparable to those of Carrel. Shell and bomb fractures almost without exception have arrived in England cases of acute or chronic osteomyelitis; of the results of prophylaxis against this terrible disease little has so far been seen. Almost the only fractures that arrive 'simple' are those that have been 'simple' uninfected from the beginning—fractures, the result of rifle and machine-gun bullets and occasionally of shrapnel balls." He admits, however, that much prophylaxis was possible under ideal conditions, but he insists that such was not attained in the vast majority of cases, and he says that there are in England and throughout the world "tens of thousands suffering the discomforts and dangers inseparable from the constantly or intermittently discharging sinus, the result of a gunshot fracture."

With this condition in mind, the author urges every surgeon to acquaint himself with the pathology of this disease, and with the treatment by which most of these sufferers can be cured. The physiology of bone and the pathology of osteomyelitis are covered. The treatment outlined is orthodox and follows long accepted lines, still the insistence upon a thorough surgical procedure is stimulating. The frequent necessity of secondary operation following the primary major attempt is recognized. The postoperative case is detailed and preference is given to the Carrel-Dakin method, without however, advice as to the minutiæ necessary for its successful use.

Although the style is involved, and renders the reading at times difficult, the book deals with a timely subject, covers it in a comprehensive way, and is well worth careful study.

A. S. I.

Infection, Immunity and Specific Therapy. By JOHN A. KOLMER, M. D. Second Edition. Cloth, $7.00. (*Philadelphia and London: W. B. Saunders Company, 1917.*)

The same statement may be made of this second edition of Kolmer's "Infection, Immunity and Specific Therapy" that was made of the first, namely, "It is the most valuable book of its kind in English Literature." Its value is now enhanced by the addition of a great deal of new and valuable material.

A brief mention of the new material with special reference to one or two subjects may suffice to give an idea of the increased value of the edition. The Schick test, which has a special field of usefulness in hospitals and institutions for the care of children is considered fully. The observation that the use of concentrated serum has lessened the incidence of serum sickness and facilitates the administration of large doses has been followed by a method, given in detail, for concentration of the serum. Very interesting are the studies by Dr. Schamberg, Dr. Raiziss and the author on the toxicity of Salvarsan.

Of equal interest is the new section on the treatment of infectious diseases, scarlet fever, poliomyelitis, pneumonia, etc., with specific serum from convalescent patients.

Among other new additions are: (1) The agglutination test in the differentiation of pneumococci; (2) the revised chapter on anaphylaxis and the special study of anaphylactic skin reaction; (3) a new method (Flexner) for the rapid production of antidysenteric serum by which effective serum for therapeutic purposes may be obtained in ten weeks.

The work may be recommended as a practical and thorough text-book on infection and immunity.

H. M. C.

Clinical Diagnosis. By CHARLES E. SIMON, B. A., M. D. Eighth Edition. Cloth.

The merits which belong to the former editions of Simon's "Clinical Diagnosis" are well known and the main interest in this volume is in the revised and new sections.

There is a new section on blood sugar with methods for its determination and one on complement fixation in the diagnosis of tuberculosis. The section on complement fixation in the diagnosis of cancer has been omitted. The author comments, "The hope that cancer might be diagnosed by this method has not been fulfilled."

The Lange colloidal gold reaction with a detailed description of the method, and with colored plates to show the reaction, has been added to this edition, as well as the new work on the determination of the existence of acidosis. The tests for blood matching have been included. There is also a detailed description of the ancestral types of various leukocytes with corresponding colored plates.

On the whole, despite the many new and changing methods in the field of clinical microscopy, the author has brought his book up to date. Methods in the old edition which have not proved valuable have been omitted and the newer ones have been added.

The colored plates are good and help to make the work a useful and valuable text-book.

H. M. C.

Plastic Surgery, Its Principles and Practice. By JOHN STAIGE DAVIS, PH. B., M. D., F. A. C. S. Instructor in Clinical Surgery, Johns Hopkins University; Assistant Visiting Surgeon, Johns Hopkins Hospital; Visiting Surgeon and Plastic Surgeon to the Union Protestant Infirmary, the Hospital for the Women of Maryland, and the Children's Hospital School, Baltimore, Md.; Fellow of the American Surgical Association, The Southern Surgical Association, etc. With 864 illustrations. Cloth, $10.00 (*Philadelphia: P. Blakiston's Son & Co., 1919.*)

The book is divided into XXV chapters, the headings of which are as follows: I. Historical Review. II. General Considerations. III. Prosthesis. IV. The Transplantation of Skin. V. The Transplantation of Other Tissues. VI. Pedunculated Flaps. VII. The Treatment of Wounds. VIII. Intractable Ulcers and Varicose Veins. IX. Scars and Keloids. X. Malformations. XI. Harelip and Cleft Palate. XII. Exstrophy of the Bladder. XIII. Epi-

spadias. XIV. Hypospadias. XV. Atresia of the Vagina. XVI. Plastic Surgery as Applied to the Various Regions. XVII. Surgery of the Eyelids. XVIII. Surgery of the Ear. XIX. Surgery of the External Nose. XX. Plastic Surgery of the Jaws, Lips and Cheeks. XXI. Surgery of the Lips. XXII. Surgery of the Cheeks. XXIII. Surgery of the Neck, Trunk and Extremities. XXIV. Surgery of the Extremities. XXV. Surgery of the Lower Extremity.

As he says in his preface, the author hopes that the book may show the general practitioner the possibilities of plastic surgery, start the student or beginner in this subject on the right track, and that in it even the more experienced surgeon may find methods with which he is unfamiliar, and which may be of use to him in dealing with plastic cases. He deplores the fact that hitherto plastic surgery has been limited to the region of the face, whereas it should include the entire surface of the body. Hence he has aimed to deal with the general principles of plastic surgery as applied to the whole body, and not the face alone.

Perhaps the key to the whole matter is to be found in the chapter on General Considerations. This deals in detail with incisions, plastic closure, needles and suture materials, methods of closing wounds without sutures, hemorrhage, drainage, dressings, massage and passive motion, etc. If the reader will study this chapter carefully before going on to those devoted to the regions, he will gain an adequate comprehension of the "little things which count for so much in the success or failure of this branch of surgery," and be better fitted to cope with the complications which arise after operative procedures.

The chapters on prosthesis, transplantation of skin, transplantation of other tissues, and pedunculated flaps, are complete in their scope and cover the ground thoroughly.

Those on the treatment of wounds, intractable ulcers and varicose veins, are carefully written, exhaustive and up-to-date. The author takes up the numerous methods of wound treatment and wound healing in a simple and clear manner, describing fully those methods which have proved to be of value, both before and during the recent great war.

The next seven chapters are devoted to malformations—congenital or acquired—such as scars and keloids, harelip and cleft palate, exstrophy of the bladder, epispadias, hypospadias, and atresia of the vagina. The last half of the book deals with plastic surgery as applied to the various regions, the different portions of the face, including the jaws, being taken up first, then the neck, trunk, and extremities. The subjects are covered very fully; the old and new operations are discussed, the author giving his own experience and modifications of the usual procedures.

Many old and new operations are described for losses of substance, the repair of defects, the restoration of function, the relief of hideous and vicious contractures, and the reconstruction of the various parts of the face and body. The illustrations are numerous, the different steps in the operative work are fully described by line drawings, or actual photographs. In addition there are exhaustive bibliographies supplied for those who desire to devote more study to any particular branch of the subject.

The author is to be congratulated on having given us the only complete and up-to-date work on plastic surgery. It cannot fail to stimulate interest in this important branch of surgery and well merits to be widely read.

W. A. F., Jr.

Microbiology: A text-book of microörganisms, general and applied. Edited by Charles E. Marshall, Professor of Microbiology and Director of Graduate School, Massachusetts Agricultural College. Cloth, $3.00. (*Philadelphia: P. Blakiston's Sons & Co., 1917.*)

This text-book, the work of numerous contributors, gives an excellent consideration of bacteriology as applied to agriculture and the special industries. There is good unity in the subject matter of the various chapters and there is little unnecessary repetition. The book is divided into three parts: The first treats of the morphology and culture; the second, of the physiology of microörganisms; the third occupies three-fourths of the book; it deals with "applied microbiology" and includes a great deal of information on the bacteriology of air, water, soil, dairy products, the microbiology of the special industries and the microbial diseases of plants, man and domestic animals. From the point of view of the non-medical student the treatment of immunity and the infectious diseases of man is quite adequate. Although the book has its most useful sphere among students of agriculture and domestic science it can be recommended to the medical student who has already acquired a knowledge of the microörganisms pathogenic for man as a means by which he may gain an insight into the development of that other branch of bacteriology to which the genius of Pasteur gave origin.

Greenspon.

PUBLICATIONS

The following eight monographs:

Free Thrombi and Ball-Thrombi in the Heart. By J. H. Hewitt, M. D. 82 pages. Price, $1.00.

Benzol as a Leucotoxin. By Laurence Selling, M. D. 60 pages. Price, $1.00.

Primary Carcinoma of the Liver. By M. C. Winternitz, M. D. 42 pages. Price 75 cents.

The Statistical Experience Data of The Johns Hopkins Hospital, Baltimore, Md., 1892-1911. By Frederick L. Hoffman, LL.D., F.S.S. 161 pages. Price, $2.00.

The Origin and Development of the Lymphatic System. By Florence R. Sabin. 94 pages. Price, $2.00.

Venous Thrombosis During Myocardial Insufficiency. By Frank J. Sladen, M. D., and Milton C. Winternitz, M. D. Price, 75 cents.

Leukaemia of the Fowl: Spontaneous and Experimental. By Harry C. Schmeisser, M. D. Price, $2.00.

The Structure of the Normal Fibers of Purkinje in the Adult Human Heart and Their Pathological Alteration in Syphilitic Myocarditis. By O. Van Der Strichit and T. Wingate Todd. Price, $2.00.

are now on sale by The Johns Hopkins Press, Baltimore. Other monographs will appear from time to time

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